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=> d his

     (FILE 'HOME' ENTERED AT 15:47:56 ON 25 FEB 2005)

     FILE 'MEDLINE, CAPLUS, SCISEARCH, BIOSIS, USPATFULL, PCTFULL' ENTERED AT
     15:48:12 ON 25 FEB 2005
L1         35567 S (FIBRONECTIN(S)(CELL(W)ADHESION)) OR (FIBRONECTIN(S)POLYMERIZ
L2           680 S (FIBRONECTIN(W)TYPE(W)(III OR 3)(W)(PEPTIDE OR POLYPEPTIDE OR
L3           426 S L1 AND L2
L4           618 S (TYPE(W)(III OR 3)(W)(REPEAT? OR DOMAIN?))(S)(COLLAGEN? OR TI
L5           529 S (BIND? OR INTERACT?)(W)(L2 OR L3)
L6            41 S (BIND? OR INTERACT?)(W)L2
L7           457 S (BIND? OR INTERACT?)(W)L4
L8          1520 S (BIND? OR INTERACT?)(W)(COLLAGEN? OR TITIN? OR TENASCIN? OR I
L9         22491 S (INHIBIT? OR DECREAS? OR BLOCK?)(W)L1
L10       135074 S (INHIBIT? OR DECREAS? OR BLOCK?)(S)((CELL?(W)ADHESION) OR FIB
L11          114 S L10 AND L1 AND L2
L12          105 DUP REM L11 (9 DUPLICATES REMOVED)
L13        34697 S (METHOD OR ASSAY OR MEANS OR PROCESS)(S)L10
L14           47 S L13 AND L3
L15           45 DUP REM L14 (2 DUPLICATES REMOVED)
L16         5833 S L10(P)FIBRONECTIN
L17           27 S L10 AND L16 AND L13 AND L3
L18           25 DUP REM L17 (2 DUPLICATES REMOVED)
L19          482 S L13(P)(UTEROGLOBIN OR UG OR CC10 OR CC16 OR CC17 OR CCSP OR B
L20           47 S L13 AND L3
L21            9 S L19 AND L3
L22            7 DUP REM L21 (2 DUPLICATES REMOVED)
L23       210167 S (ASSAY OR METHOD OR PROCESS)(S)(IDENTIFY? OR SCREEN? OR EVALU
L24         2228 S L23(P)(L1 OR L2)
L25         4627 S L23(P)L10
L26          716 S L24 AND L25
L27         3702 S L23(P)FIBRONECTIN
L28          886 S L27(P)(L10)
L29        22499 S (L9 OR L10)(P)(L1 OR L2)
L30          442 S L27 AND L13 AND L10
L31           35 S L11 AND L27
L32           35 DUP REM L31 (0 DUPLICATES REMOVED)
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=> s fibronectin(s)(cell?(w)adhesion)
   4 FILES SEARCHED...
L1       10002 FIBRONECTIN(S)(CELL?(W) ADHESION)

=> s (inhibit? or decreas? or block?)(s)l1
L2        3468 (INHIBIT? OR DECREAS? OR BLOCK?)(S) L1

=> s (method or assay? or process?)(s)(identify? or detect? or evaluat?)(s)(compound? or
polypeptide? or protein? or molecule?)(s)(interact? or bind? or associat? or
complex?)(s)fibronectin
   3 FILES SEARCHED...
   5 FILES SEARCHED...
L3         935 (METHOD OR ASSAY? OR PROCESS?)(S)(IDENTIFY? OR DETECT? OR EVALUA
               T?)(S)(COMPOUND? OR POLYPEPTIDE? OR PROTEIN? OR MOLECULE?)(S)(IN
               TERACT? OR BIND? OR ASSOCIAT? OR COMPLEX?)(S) FIBRONECTIN

=> s l3 and l2
L4         138 L3 AND L2

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5          138 DUP REM L4 (0 DUPLICATES REMOVED)

=> s l5 and py<=2001
   1 FILES SEARCHED...
   3 FILES SEARCHED...
L6          67 L5 AND PY<=2001

=> d ibib abs 1-67

L6   ANSWER 1 OF 67  SCISEARCH  COPYRIGHT (c) 2005 The Thomson Corporation.  on
     STN
ACCESSION NUMBER:    2001:980387  SCISEARCH
THE GENUINE ARTICLE: 499XG
TITLE:               NG2/HMPG modulation of human articular chondrocyte
                     adhesion to type VI collagen is lost in osteoarthritis
AUTHOR:              Midwood K S; Salter D M (Reprint)
CORPORATE SOURCE:    Univ Edinburgh, Sch Med, Dept Pathol, Teviot Pl, Edinburgh
                     EH8 9AG, Midlothian, Scotland (Reprint); Univ Edinburgh,
                     Sch Med, Dept Pathol, Edinburgh EH8 9AG, Midlothian,
                     Scotland
COUNTRY OF AUTHOR:   Scotland
SOURCE:              JOURNAL OF PATHOLOGY, (DEC 2001) Vol. 195, No.
                     5, pp. 631-635.
                     Publisher: JOHN WILEY & SONS LTD, BAFFINS LANE CHICHESTER,
                     W SUSSEX PO19 1UD, ENGLAND.
                     ISSN: 0022-3417.
DOCUMENT TYPE:       Article; Journal
LANGUAGE:            English
REFERENCE COUNT:     28
                    *ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*
AB       NG2/human melanoma proteoglycan (HMPG) is a chondroitin sulphate
     proteoglycan (CSPG),. expressed by chondrocytes in fetal and in normal and
     osteoarthritic (OA) adult articular cartilage. NG2/HMPG is a receptor for
     extracellular matrix proteins, including type VI collagen, and
     regulates fill integrin binding to fibronectin. This
     study was undertaken to identify whether NG2/HMPG had similar
     activities in human articular chondrocytes (HACs). Normal and OA adult HAC
     adhesion to fibronectin, type II or type VI collagen was
     assessed using a methylene blue assay. The requirement for
     integrins, NG2/HMPG, and integrin-associated signalling
     molecules was investigated using anti-beta1 integrin and anti-HMPG
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antibodies and pharmacological **inhibitors** of signalling **molecules**. The adhesion of normal and OA HACs to **fibronectin**, type II and type VI collagen was beta1 integrin-dependent. Normal HAC adhesion to type V1 collagen was stimulated by anti-HMPG antibodies. This effect was **inhibited** by pertussis toxin. Anti-HMPG antibodies had no effect on OA chondrocyte adhesion to type VI collagen, or on normal and OA **cell adhesion** to **fibronectin** and type II collagen. The results show that NG2/HMPG modulates integrin-mediated **interactions** of normal HACs with type VI collagen. Loss of this activity may be of importance in the progression of osteoarthritis. Copyright (C) 2001 John Wiley & Sons, Ltd.

L6 ANSWER 2 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 2001:219299 SCISEARCH
THE GENUINE ARTICLE: 409JV
TITLE: Focal adhesion kinase (FAK) phosphorylation is not required for genistein-induced FAK-beta-1-integrin complex formation
AUTHOR: Liu Y Q; Kyle E; Lieberman R; Crowell J; Kelloff G; Bergan R C (Reprint)
CORPORATE SOURCE: Northwestern Univ, Sch Med, Dept Med, Div Hematol Oncol, Olson 8524, 710 N Fairbanks, Chicago, IL 60611 USA (Reprint); Northwestern Univ, Sch Med, Dept Med, Div Hematol Oncol, Chicago, IL 60611 USA; Northwestern Univ, Robert H Lurie Canc Ctr, Chicago, IL 60611 USA; NCI, Bethesda, MD 20892 USA
COUNTRY OF AUTHOR: USA
SOURCE: CLINICAL & EXPERIMENTAL METASTASIS, (**21 FEB 2000**) Vol. 18, No. 3, pp. 203-212.
Publisher: KLUWER ACADEMIC PUBL, SPUIBOULEVARD 50, PO BOX 17, 3300 AA DORDRECHT, NETHERLANDS.
ISSN: 0262-0898.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 41

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB It has previously been shown that changes in the activity of focal adhesion kinase (FAK), and its **binding** to beta -1-integrin, accompany genistein-induced adhesion of prostate cells. Consumption of genistein world wide is **associated** with a lower incidence of metastatic prostate cancer. Early human clinical trials of genistein are under way to **evaluate** genistein's potential causal role in this regard. Though an important **cell adhesion-associated** signaling **molecule**, FAK's role in regulating prostate **cell adhesion** was not clear. Elucidation of this **process** would provide important information relating to both biology and potential clinical endpoints. It was hypothesized that FAK activation and **complex** formation are temporally related in prostate cells, and can thus be separated. Significant activation of FAK was demonstrated when cells adhered to **fibronectin**, as compared to poly-l-lysine, thus demonstrating that beta -1-integrin plays a significant role in activating FAK. Neither FAK activation, nor FAK-integrin **complex** formation, required beta -1-integrin ligand. However, disruption of the cellular cytoskeleton by cytochalasin D prevented FAK activation, but did not **block** genistein-induced **complex** formation. In the face of a disrupted cytoskeleton, signaling through FAK could not be restored through either integrin cross linking, or re-establishment of tensile forces via attachment to solid matrix. These studies demonstrate that FAK-beta -1-integrin **complex** formation does not require FAK activation, suggesting that it is an early event in prostate **cell adhesion**. An

intact cytoskeleton is necessary for FAK activation. The functional importance of beta -1-integrin in prostate cells is demonstrated. Current findings support plans to test genistein in prostate cancer.

L6 ANSWER 3 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2001:115641 SCISEARCH
THE GENUINE ARTICLE: 396XA
TITLE: Differential inhibition of renal cancer cell invasion mediated by fibronectin, collagen IV and laminin
AUTHOR: Brenner W (Reprint); Gross S; Steinbach F; Horn S; Hohenfellner R; Thuroff J W
CORPORATE SOURCE: Univ Mainz, Urol Clin & Policlin, Langenbeckstr 1, D-55131 Mainz, Germany (Reprint); Univ Mainz, Urol Clin & Policlin, D-55131 Mainz, Germany; Univ Magdeburg, Urol Clin & Policlin, D-39106 Magdeburg, Germany; Univ Mainz, Med Clin & Policlin 3, D-6500 Mainz, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: CANCER LETTERS, (**31 JUL 2000**) Vol. 155, No. 2, pp. 199-205.
Publisher: ELSEVIER SCI IRELAND LTD, CUSTOMER RELATIONS MANAGER, BAY 15, SHANNON INDUSTRIAL ESTATE CO, CLARE, IRELAND.
ISSN: 0304-3835.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 17
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Invasion of tumor cells into the extracellular matrix is an essential step in the formation of metastases in renal cancer. **Cell adhesion molecules** such as beta (1)-integrins, which **bind** to the RGD sequence (arginine-glycine-asparagine) and CD44 are involved in this **process**. We examined the invasion of a renal carcinoma cell line (CCF-RC1) into the extracellular matrix **compounds fibronectin**. collagen IV and laminin and the effect of TGF beta and IFN gamma on this **process**. The **inhibitory** effect of an antibody against the beta (1)-subunit of integrins (CD29), as well as a pentapeptide including the RGD sequence, was also **evaluated**. A micro-chemotaxis chamber, including a polycarbonate membrane with a pore diameter of 8 mum, was used for quantification of cell migration. The addition of the extracellular matrix **compounds fibronectin**, laminin and collagen IV resulted in a 5-10-fold increase in invasion. This increased invasion depends strongly on the presence of beta (1)-integrins, shown by the use of an antibody against CD29 or a RGD including peptide which **inhibit** the cell migration by approximately 88%. CD44 is less involved in collagen IV dependent migration and almost no influence of CD44 was observed on a **fibronectin** and laminin dependent migration. TNF alpha and IFN gamma did not significantly influence the expression of CD29 or CD44, and no alteration in tumor cell migration was observed. These results show that the invasion of renal cancer cells is differentially regulated by **compounds** of the extracellular matrix, whereby **fibronectin** seems to be the most critical factor. The molecular **interactions** in this **process** are strongly dependent on beta (1)-integrins and the corresponding amino acid sequence ROD.

L6 ANSWER 4 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2000:943926 SCISEARCH
THE GENUINE ARTICLE: 381AH
TITLE: Cell adhesion and focal adhesion kinase regulate insulin

receptor substrate-1 expression
AUTHOR: Lebrun P; Baron V (Reprint); Hauck C R; Schlaepfer D D; VanObberghen E
CORPORATE SOURCE: INSERM, U145, INST FEDERATIF RECH, 50 AVE VALOMBROSE, F-06107 NICE 2, FRANCE (Reprint); INSERM, U145, INST FEDERATIF RECH, F-06107 NICE 2, FRANCE; SCRIPPS RES INST, DEPT IMMUNOL, LA JOLLA, CA 92037
COUNTRY OF AUTHOR: FRANCE; USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (**8 DEC 2000**) Vol. 275, No. 49, pp. 38371-38377.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 51
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Integrins are transmembrane receptors involved in **interactions** between cells and extracellular matrix **proteins**. Here we show that **cell adhesion** regulates insulin receptor substrate-1 (IRS-1) mRNA synthesis, When fibroblasts are held in suspension, lower levels of IRS-1 mRNA, but not of IRS-2 mRNA, are **detected**, and this effect is due to the negative regulation of IRS-1 transcription rather than to **decreased** mRNA stability. Upon **fibronectin**- or vitronectin-mediated integrin stimulation, the level of IRS-1 mRNA was restored within 4 h, The focal adhesion kinase (FAK) is known to be activated upon integrin stimulation, and we found that IRS-1 was not expressed in FAK(-/-) cells. Stable re-expression of epitope-tagged FAK in FAK(-/-) fibroblasts (DA2 cells) restored normal levels of IRS-1 expression, confirming that IRS-1 mRNA expression is regulated by FAK, It is known that integrins activate the JNK pathway. However, in adherent FAK(-/-) cells, we failed to **detect** activation of JNK, whereas JNK was stimulated in DA2 cells, This confirms the role of FAK in integrin-induced JNK stimulation. FAR-independent stimulation of JNK with anisomycin treatment both in FAK(-/-) cells and in suspended FAK(-/-) cells confirmed that IRS-1 mRNA transcription can be partially regulated by JNK, We suggest that integrins can modulate insulin and insulin-like growth factor-1 signaling pathways by regulating the levels of IRS-1 in cells and that FAR-mediated signaling to JNK is one pathway involved in this **process**.

L6 ANSWER 5 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2000:763458 SCISEARCH
THE GENUINE ARTICLE: 359ZG
TITLE: Destruction or bile ducts in primary biliary cirrhosis
AUTHOR: Nakanuma Y (Reprint); Tsuneyama K; Sasaki M; Harada K
CORPORATE SOURCE: KANAZAWA UNIV, SCH MED, DEPT PATHOL 2, KANAZAWA, ISHIKAWA 920864, JAPAN (Reprint)
COUNTRY OF AUTHOR: JAPAN
SOURCE: BEST PRACTICE & RESEARCH IN CLINICAL GASTROENTEROLOGY, (**AUG 2000**) Vol. 14, No. 4, pp. 549-570.
Publisher: BAILLIERE TINDALL, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND.
ISSN: 1521-6918.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: CLIN
LANGUAGE: English
REFERENCE COUNT: 83
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Primary biliary cirrhosis is characterized by the immune-mediated,

progressive destruction of interlobular bile ducts. Lymphoid cells migrate into the biliary epithelial layer through integrin alpha(4)/ **fibronectin interaction** and are responsible for chronic destructive cholangitis. The bile ducts express intercellular adhesion **molecule**-1 (ICAM-1) and vascular **cellular adhesion molecule**-1 (ICAM-1), and infiltrating lymphocytes express LFA1 and VLA4, facilitating their **interaction** . Epithelioid granulomas contain foamy cells ingesting biliary lipids, and CD1d was **detectable** in epithelioid granulomas, suggesting that the biliary substance(s) which are leaked is a trigger for chronic destructive cholangitis. Apoptotic biliary destruction is brought about by antigen-specific and non-specific reactions. Shrunken biliary epithelial cells with pyknotic nuclei positive for terminal deoxynucleotidyl transferase-mediated deoxyuridine triphosphate nick-end labelling (TUNEL) may reflect apoptotic **processes**. Increased expression of caspase-3 and -8 with DNA fragmentation factor on the bile ducts may reflect molecular events during apoptosis, and down-regulation of Bcl-2 of biliary epithelial cells seems to facilitate apoptosis. Multiple factors, particularly the Fas system, are stimuli of apoptosis. Anoikis with **decreased** biliary expression of integrin 6, a ligand for laminin, may also be involved in biliary epithelial apoptosis.

L6 ANSWER 6 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 2000:395902 SCISEARCH
THE GENUINE ARTICLE: 315WQ
TITLE: Signal transduction mediated by adhesion of human trabecular meshwork cells to extracellular matrix
AUTHOR: Zhou L L; Cheng E L L; Rege P; Yue B Y J T (Reprint)
CORPORATE SOURCE: UNIV ILLINOIS, COLL MED, DEPT OPHTHALMOL & VISUAL SCI, 1855 W TAYLOR ST, CHICAGO, IL 60680 (Reprint); UNIV ILLINOIS, COLL MED, DEPT OPHTHALMOL & VISUAL SCI, CHICAGO, IL 60680
COUNTRY OF AUTHOR: USA
SOURCE: EXPERIMENTAL EYE RESEARCH, (**APR 2000**) Vol. 70, No. 4, pp. 457-465.
Publisher: ACADEMIC PRESS LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND.
ISSN: 0014-4835.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 56

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB In this study we investigated the signaling event induced by adhesion of human trabecular meshwork (TM) cells to extracellular matrix (ECM) elements such as **fibronectin**, The role of tyrosine phosphorylation in adhesion was **evaluated**. A number of intracellular entities involved in the adhesion-mediated pathways were identified, For the experiments, human TM cells were seeded onto **fibronectin**- or polylysine (negative control)-coated plates. Fifteen, 30, 90 and 240 min after the seeding, cell lysates were collected. Immunoblotting analysis revealed that tyrosine phosphorylation occurred within 15 min of adhesion of TM cells to **fibronectin** and the level increased with time. The phosphotyrosyl **proteins** had molecular masses 25-220 kDa. A much lower level of tyrosine phosphorylation was observed when cells were plated on polylysine. Immunoprecipitation experiments indicated that the phosphotyrosine-containing **proteins** included focal adhesion kinase, paxillin, phosphatidylinositol 3-kinase and mitogen activated **protein** kinase. Within 30 ruin of adherence to **fibronectin**, human TM cells immunostained for paxillin and phosphotyrosine and exhibited

prominent focal contacts. When treated with tyrosine kinase **inhibitors** genistein and herbimycin A and a **protein** kinase C (PRC) pseudosubstrate peptide **inhibitor, cell adhesion** to **fibronectin** was compromised and focal contact formation was limited. These results demonstrated that in human TM cells, tyrosine kinase was activated upon their adherence to **fibronectin**. PKC also appeared to play a role in modulation of the cell-matrix adhesion **process**. The current study provides insight into the signaling pathways that are linked to the ECM-induced events in TM cells. Elucidation of the hierarchy of signal responses may help develop strategies manipulating the cell-matrix **interactions** in the TM system. (C) 2000 Academic Press.

L6 ANSWER 7 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1998:816022 SCISEARCH
THE GENUINE ARTICLE: 130BL
TITLE: Differential expression and distribution of focal adhesion and cell adhesion molecules in rat hepatocyte differentiation
AUTHOR: Kim T H; Bowen W C; Stolz D B; Runge D; Mars W M; Michalopoulos G K (Reprint)
CORPORATE SOURCE: UNIV PITTSBURGH, SCH MED, DEPT PATHOL, PITTSBURGH, PA 15261 (Reprint); UNIV PITTSBURGH, SCH MED, DEPT PATHOL, PITTSBURGH, PA 15261; UNIV PITTSBURGH, SCH MED, DEPT CELL BIOL, PITTSBURGH, PA 15261
COUNTRY OF AUTHOR: USA
SOURCE: EXPERIMENTAL CELL RESEARCH, (**10 OCT 1998**) Vol. 244, No. 1, pp. 93-104.
Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0014-4827.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 51

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Hepatocytes in primary culture enter into clonal proliferation in the chemically defined hepatocyte growth medium in the presence of hepatocyte growth factor and epidermal growth factor. Hepatocyte proliferation is **associated** with loss of differentiated gene expression. Overlay of matrix derived from Engelbreth-Holm-Swarm mouse sarcoma (Matrigel) on proliferating hepatocytes Hepatocytes induces reexpression of the hepatic differentiation marker genes. To explore the role of matrix in the differentiation **process** of hepatocytes, we examined the mRNAs of **fibronectin**, vitronectin, and entactin in proliferating hepatocytes and Matrigel-treated hepatocytes. **Fibronectin** mRNA increased in proliferating hepatocytes at days 2-10 and then **decreased**; however, vitronectin mRNA disappeared in proliferating hepatocytes and was reexpressed in Matrigel-treated hepatocytes. We also found that focal adhesion kinase and paxillin were strongly increased in Matrigel-treated hepatocytes, and E-cadherin and beta-catenin slightly increased in Matrigel-treated hepatocytes, suggesting that both cell-to extracellular matrix and cell-to-cell **interactions** may be an essential part of hepatocyte differentiation. To **evaluate** the distribution of focal adhesion **associated molecules** and cell-to-**cell adhesion molecules**, Triton X-100 soluble and insoluble fractions were examined at days 8, 9, 10, and 11 in proliferating hepatocytes and Matrigel-treated cells. We found that E-cadherin in Triton X-100 insoluble fractions dramatically **decreased** in Matrigel-treated hepatocytes; however, beta-catenin strongly increased in Triton X-100 soluble fractions of Matrigel-treated

hepatocytes. These results suggest that the distribution of both focal adhesion **associated molecules** and **cell adhesion molecules** are reorganized during the **process** of differentiation induced by overlay of Matrigel. (C) 1998 Academic Press.

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ACCESSION NUMBER: 1998:720659 SCISEARCH
THE GENUINE ARTICLE: 119EP
TITLE: Characterization of the macromolecular components of the articular cartilage surface
AUTHOR: Noyori K; Takagi T; Jasin H E (Reprint)
CORPORATE SOURCE: UNIV ARKANSAS MED SCI, THERESA SCHEU RHEUMATOID ARTHRIT RES LAB, DEPT INTERNAL MED, MAIL SLOT 509, LITTLE ROCK, AR 72205 (Reprint); UNIV ARKANSAS MED SCI, THERESA SCHEU RHEUMATOID ARTHRIT RES LAB, DEPT INTERNAL MED, LITTLE ROCK, AR 72205; JOHN L MCCLELLAN MEM VET ADM MED CTR, LITTLE ROCK, AR 72205; YOKOHAMA CITY UNIV, SCH MED, DEPT ORTHOPAED SURG, YOKOHAMA, KANAGAWA 232, JAPAN
COUNTRY OF AUTHOR: USA; JAPAN
SOURCE: RHEUMATOLOGY INTERNATIONAL, (**AUG 1998**) Vol. 18, No. 2, pp. 71-77.
Publisher: SPRINGER VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010.
ISSN: 0172-8172.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 28

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The intact surface of articular cartilage is a highly organized structure composed of a variety of macromolecules. The studies reported here deal with a partial characterization of the non-covalently bound components of the outermost layer of articular cartilage. Normal bovine and human cartilage articular surfaces were extracted for 5 min with 4-M guanidine HCl solution. Analysis and quantitation of small proteoglycans in the extract were carried out by PAGE (polyacrylamide gel electrophoresis), Western blot, and radioimmunoassays. The present studies indicate that the major **proteins** extracted from the articular surface of bovine and human cartilage are the collagen-**binding** small proteoglycans designated as fibromodulin and albumin. **Fibronectin**, decorin, and biglycan were also **detected** in smaller amounts. Immunoblotting of the surface material developed with a monoclonal antibody with keratan sulfate specificity confirmed the presence of fibromodulin coinciding with the major **protein** band of approximately 70-100-kDa molecular mass. Gel filtration chromatography of the surface material confirmed the previous results. Additional in vitro **assays** showed that the collagen-**binding** material extracted from the cartilage surface, contained the small proteoglycans. Anti-human fibromodulin antibodies bound in significantly greater amounts to the intact articular surfaces than to cut surfaces of normal human cartilage. It is concluded that small, non-aggregating proteoglycans constitute the major proteoglycan species non-covalently bound to macromolecules at the articular surface of cartilage partially responsible for the interference of anti-collagen type II antibody **binding** and for the **inhibition** of **cell adhesion** to the intact surface.

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ACCESSION NUMBER: 1998:669864 SCISEARCH

THE GENUINE ARTICLE: 114YV
TITLE: Adhesive interactions of human multiple myeloma cell lines with different extracellular matrix molecules
AUTHOR: Kibler C; Schermutzki F; Waller H D; Timpl R; Muller C A; Klein G (Reprint)
CORPORATE SOURCE: UNIV TUBINGEN, MED CLIN, DEPT 2, SECT TRANSPLANTAT IMMUNOL & IMMUNOHEMATOL, D-72076 TUBINGEN, GERMANY (Reprint); UNIV TUBINGEN, MED CLIN, DEPT 2, SECT TRANSPLANTAT IMMUNOL & IMMUNOHEMATOL, D-72076 TUBINGEN, GERMANY; MAX PLANCK INST BIOCHEM, D-82152 MARTINSRIED, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: CELL ADHESION AND COMMUNICATION, (**AUG 1998**) Vol. 5, No. 4, pp. 307-323.
Publisher: HARWOOD ACAD PUBL GMBH, C/O STBS LTD, PO BOX 90, READING RG1 8JL, BERKS, ENGLAND.
ISSN: 1061-5385.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 52

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Multiple myeloma represents a human B cell malignancy which is characterized by a predominant localization of the malignant cell clone within the bone marrow. With the exception of the terminal stage of the disease the myeloma tumor cells do not circulate in the peripheral blood. The bone marrow microenvironment is believed to play an important role in homing, proliferation and terminal differentiation of myeloma cells. Here we have studied the expression of several extracellular matrix (ECM) **molecules** in the bone marrow of multiple myeloma patients and analyzed their adhesive capacities with four different human myeloma-derived cell lines. All ECM **molecules** analyzed (tenascin, laminin, **fibronectin**, collagen types I, III, V and VT) could be **detected** in bone marrow cryostat sections of multiple myeloma patients. Adhesion : **assays** showed that only laminin, the microfibrillar collagen type VI and **fibronectin** were strong adhesive components for the myeloma cell lines U266, IM-9, OPM-2 and NCI-H929. Tenascin and collagen type I were only weak adhesive substrates for these myeloma **cells**. **Adhesion** to laminin and **fibronectin** was beta 1-integrin-mediated since addition of anti-beta 1-integrin antibodies could **inhibit** the **binding** of the four different cell types to both matrix **molecules**. In contrast, integrins do not seem to be involved in **binding** of the myeloma cells to collagen type VI. Instead, **inhibition** of **binding** by heparin suggested that membrane-bound heparan sulfate proteoglycans are responsible ligands for **binding** to collagen type VI. Adhesion **assays** with several B-cell lines resembling earlier differentiation stages revealed only weak **interactions** with tenascin and no **interactions** with collagen type VI, laminin or **fibronectin**. In summary, the **interactions** of human myeloma cells with the extracellular matrix may explain the specific retention of the plasma cells within the bone marrow.

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ACCESSION NUMBER: 1998:487207 SCISEARCH
THE GENUINE ARTICLE: ZV178
TITLE: Hormonal regulation of focal adhesions in bovine adrenocortical cells: induction of paxillin dephosphorylation by adrenocorticotropic hormone
AUTHOR: Vilgrain I (Reprint); Chinn A; Gaillard I; Chambaz E M; Feige J J

CORPORATE SOURCE: CEA, DEPT BIOL MOL & STRUCT, INSERM, U244, 17 RUE MARTYRS, F-38054 GRENOBLE 9, FRANCE (Reprint)
COUNTRY OF AUTHOR: FRANCE
SOURCE: BIOCHEMICAL JOURNAL, (**1 JUN 1998**) Vol. 332, Part 2, pp. 533-540.
Publisher: PORTLAND PRESS, 59 PORTLAND PLACE, LONDON W1N 3AJ, ENGLAND.
ISSN: 0264-6021.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 46
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A study of bovine adrenocortical cell shape on adrenocorticotropic hormone (ACTH) challenge showed that the cells round up and develop arborized **processes**. This effect was found to be (1) specific for ACTH because angiotensin II and basic fibroblast growth factor have no effect; (2) mediated by a cAMP-dependent pathway because forskolin reproduces the effect of the hormone; (3) **inhibited** by sodium orthovanadate, a phosphotyrosine phosphatase **inhibitor**, but unchanged by okadaic acid, a serine/threonine phosphatase **inhibitor**; and (4) correlated with a complete loss of focal adhesions. Biochemical studies of the focal-adhesion-**associated proteins** showed that pp125(fak), vinculin (110 kDa) and paxillin (70 kDa) were **detected** in the Triton X-100-insoluble fraction from adrenocortical cells. During **cell adhesion** on **fibronectin** as substratum, two major phosphotyrosine-containing **proteins** of molecular masses 125 and 68 kDa were immunodetected in the same fraction. A dramatic **decrease** in the extent of tyrosine phosphorylation of these **proteins** was observed within 60 min after treatment with ACTH. No change in pp125(fak) tyrosine phosphorylation nor in Src activity was **detected**. In contrast, paxillin was found to be tyrosine-dephosphorylated in a time-dependent manner in ACTH-treated cells. Sodium orthovanadate completely prevented the effect of ACTH. These observations suggest a possible role for phosphotyrosine phosphatases in hormone-dependent cellular regulatory **processes**.

L6 ANSWER 11 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1998:254129 SCISEARCH
THE GENUINE ARTICLE: ZD515
TITLE: Mac-2 binding protein is a cell-adhesive protein of the extracellular matrix which self-assembles into ring-like structures and binds beta 1 integrins, collagens and fibronectin
AUTHOR: Sasaki T; Brakebusch C; Engel J; Timpl R (Reprint)
CORPORATE SOURCE: MAX PLANCK INST BIOCHEM, D-82152 MARTINSRIED, GERMANY (Reprint); MAX PLANCK INST BIOCHEM, D-82152 MARTINSRIED, GERMANY; UNIV BASEL, BIOZENTRUM, CH-4056 BASEL, SWITZERLAND
COUNTRY OF AUTHOR: GERMANY; SWITZERLAND
SOURCE: EMBO JOURNAL, (**16 MAR 1998**) Vol. 17, No. 6, pp. 1606-1613.
Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD, ENGLAND OX2 6DP.
ISSN: 0261-4189.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 45
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Human Mac-2 **binding protein** (M2BP) was prepared in recombinant form from the culture medium of 293 kidney cells and consisted of a 92 kDa subunit, The **protein** was obtained in a native state as indicated by CD spectroscopy, demonstrating alpha-helical and beta-type structure, and by protease resistance and immunological analysis. It was highly modified by N- and O-glycosylation but not by glycosaminoglycans. Ultracentrifugation showed non-covalent **association** into oligomers with molar masses of 1000-1500 kDa. Electron microscopy showed ring-like shapes with diameters of 30-40 nm. M2BP bound in solid-phase **assays** to collagens IV: V and VI, **fibronectin** and nidogen, but not to fibrillar collagens I and III or other basement membrane **proteins**. The **protein** also mediated adhesion of cell lines at comparable strength with laminin. Adhesion to M2BP was **inhibited** by antibodies to integrin beta 1 subunits but not to alpha 2 and alpha 6 subunits, RGD peptide or lactose. This distinguishes **cell adhesion** of M2BP from that of laminin and excludes involvement of lactose-**binding** galectin-3. Immunological **assays** demonstrated variable secretion by cultured human cells of M2BP, which was **detected** in the extracellular matrix of several mouse tissues.

L6 ANSWER 12 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 1998:128941 SCISEARCH
THE GENUINE ARTICLE: YV635
TITLE: Adhesion of activated platelets to endothelial cells: Evidence for a GPIIbIIIa-dependent bridging mechanism and novel roles for endothelial intercellular adhesion molecule 1 (ICAM-1), alpha(v)beta(3) integrin, and GPIb alpha
AUTHOR: Bombeli T; Schwartz B R; Harlan J M (Reprint)
CORPORATE SOURCE: UNIV WASHINGTON, DIV HEMATOL, BOX 357710, 1959 PACIFIC ST NE, SEATTLE, WA 98195 (Reprint); UNIV WASHINGTON, DIV HEMATOL, SEATTLE, WA 98195
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (**2 FEB 1998**) Vol. 187, No. 3, pp. 329-339.
Publisher: ROCKEFELLER UNIV PRESS, 1114 FIRST AVE, 4TH FL, NEW YORK, NY 10021.
ISSN: 0022-1007.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 51

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Although it has been reported that activated platelets can adhere to intact endothelium, the receptors involved have not been fully characterized. Also, it is not clear whether activated platelets **bind** primarily to matrix **proteins** at sites of endothelial cell denudation or directly to endothelial cells. Thus, this study was designed to further clarify the mechanisms of activated platelet adhesion to endothelium. Unstimulated human umbilical vein endothelial cell (HUVEC) monolayers were incubated with washed, stained, and thrombin-activated human platelets. To exclude matrix involvement, HUVEC were harvested mechanically and platelet **binding** was measured by now cytometry. Before the adhesion **assay**, platelets or HUVEC were treated with different receptor antagonists. Whereas **blockade** of platelet beta(1) integrins, GPIb alpha, GPIV, P-selectin, and platelet-endothelial **cell adhesion molecule** (PECAM)-1 did not reduce platelet adhesion to HUVEC, **blockade** of platelet GPIIbIIIa by antibodies or Arg-Gly-Asp (RGD) peptides markedly **decreased** adhesion. Moreover, when platelets were treated with

blocking antibodies to GPIIbIIIa-**binding** adhesive **proteins**, including fibrinogen and **fibronectin**, and von Willebrand factor (vWF), platelet **binding** was also reduced markedly. Addition of fibrinogen, **fibronectin**, or vWF further increased platelet adhesion, indicating that both endogenous platelet-exposed and exogenous adhesive **proteins** can participate in the **binding process**. **Evaluation** of the HUVEC receptors revealed predominant involvement of intercellular adhesion **molecule** (ICAM)-1 and alpha(v) beta(3) integrin. **Blockade** of these two receptors by antibodies **decreased** platelet **binding** significantly. Also, there was evidence that a component of platelet adhesion was mediated by endothelial GPIb alpha. **Blockade** of beta(1) integrins, E-selectin, P-selectin, PECAM-1, vascular **cell adhesion molecule** (VCAM)-1 and different matrix **proteins** on HUVEC did not affect platelet adhesion. In conclusion, we show that activated platelet **binding** to HUVEC monolayers is mediated by a GPIIbIIIa-dependent bridging mechanism involving platelet-bound adhesive **proteins** and the endothelial cell receptors ICAM-1, alpha(v) beta(3) integrin, and, to a lesser extent, GPIb alpha.

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ACCESSION NUMBER: 1998:695 SCISEARCH
THE GENUINE ARTICLE: YK702
TITLE: Antagonism of the GPIIb/IIIa receptor with the nonpeptidic molecule BIBU52: Inhibition of platelet aggregation in vitro and antithrombotic efficacy in vivo
AUTHOR: Guth B D (Reprint); SeewaldtBecker E; Himmelsbach F; Weisenberger H; Muller T H
CORPORATE SOURCE: DR KARL THOMAE GMBH, DEPT BIOL RES, POSTFACH 1755, D-88397 BIBERACH, GERMANY (Reprint); DR KARL THOMAE GMBH, DEPT CHEM RES, D-88397 BIBERACH, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: JOURNAL OF CARDIOVASCULAR PHARMACOLOGY, **(AUG 1997***)**
Vol. 30, No. 2, pp. 261-272.
Publisher: LIPPINCOTT-RAVEN PUBL, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106.
ISSN: 0160-2446.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 28

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB **The glycoprotein (GP) IIb/IIIa (the alpha(IIb)beta(3) integrin) found on platelets ***binds** fibrinogen or von Willebrand factor when the platelet is activated, thereby mediating the aggregation of platelets. **Blockade** of the GPIIb/IIIa should prevent platelet aggregation independent of the substance or substances responsible for activating the platelets. This comprehensive **inhibition** of platelet aggregation is thought to be an effective therapeutic approach to various clinical thromboembolic syndromes. This study investigated the platelet **inhibition** provided by **blocking** GPIIb/IIIa by using a new nonpeptidic **molecule**, BIBU52, in both in vitro and in vivo models. BIBU52 competes with [I-125]fibrinogen for **binding** sites on human platelets in a Ca2+ and pH-dependent manner with a 50% **inhibitory** concentration (IC50) of 35 +/- 12 nM. BIBU52 **inhibited** the aggregation of human platelets in platelet-rich plasma induced by collagen (1-2 mu g/ml), adenosine diphosphate (ADP; 2.5 mu M), and a thrombin receptor-activating peptide (TRAP; SFLLRNPNDKYEPF-NH2; 25 mu M) with IC50 values of 82, 83, and 200 nM, respectively. The **inhibition** of platelet aggregation by BIBU52

was found to be highly species dependent. BIBU52 **inhibited** aggregation in plasma from rhesus and marmoset monkeys with an IC50 Of 150 nM but was totally ineffective in rat plasma. The selectivity of BIBU52 for **inhibiting** GPIIb/IIIa in comparison with other adhesion **molecules** was investigated in a human endothelial **cell adhesion assay**. The adhesion of human endothelial cells to matrices of vitronectin, **fibronectin**, collagen I, or laminin was not affected by concentrations as high as 100 mu M BIBU52; thus BIBU52 demonstrates a high selectivity for the human GPIIb/IIIa. The antithrombotic effect of BIBU52 in vivo was investigated in three animal models of recurrent arterial thrombus formation. In the guinea pig aorta, BIBU52 **inhibited** thrombus formation dose dependently, with lack of thrombus formation for 1 h after a bolus dose of 1.0 mg/kg i.v.. Both acetylsalicylic acid and dazoxiben were less effective in this model. In pigs with recurrent thrombus formation in the carotid artery, 1.0 mg/kg i.v. also **inhibited** thrombus formation. Heparin was not effective in the pig, and acetylsalicylic acid was only partially effective. In the pig, the dose of 1.0 mg/kg i.v. BIBU52 also was **associated** with a 70% **inhibition** of collagen-induced platelet aggregation ex vivo but with only a transient prolongation of sublingual bleeding time to a maximum of 2.5-fold and without other hemodynamic effects. In the marmoset monkey, a dose of 10 mu g/kg i.v. could abolish recurrent arterial thrombosis. Hemodynamic effects of BIBU52 in anesthetized pigs were not **detected** in doses less than or equal to 10 mg/kg. These data demonstrate that BIBU52 is a potent and selective antagonist of the human GPIIb/IIIa receptor and capable of substantial **inhibition** of platelet aggregation in vitro and ex vivo as well as **inhibition** of arterial thrombus formation in vivo in animal models of thrombosis.

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ACCESSION NUMBER: 97:625643 SCISEARCH
THE GENUINE ARTICLE: XR357
TITLE: Expression of the integrin alpha 5 subunit and its mediated cell adhesion in hepatocellular carcinoma
AUTHOR: Yao M; Zhou X D (Reprint); Zha X L; Shi D R; Fu J; He J Y; Lu H F; Tang Z Y
CORPORATE SOURCE: SHANGHAI MED UNIV, ZHONG SHAN HOSP, LIVER CANC INST, SHANGHAI 200032, PEOPLES R CHINA (Reprint); SHANGHAI MED UNIV, ZHONG SHAN HOSP, LIVER CANC INST, SHANGHAI 200032, PEOPLES R CHINA; SHANGHAI MED UNIV, FAC BASIC MED SCI, DEPT BIOCHEM, SHANGHAI 200032, PEOPLES R CHINA; SHANGHAI MED UNIV, TUMOR HOSP, DEPT PATHOL, SHANGHAI 200032, PEOPLES R CHINA
COUNTRY OF AUTHOR: PEOPLES R CHINA
SOURCE: JOURNAL OF CANCER RESEARCH AND CLINICAL ONCOLOGY, (**AUG 1997**) Vol. 123, No. 8, pp. 435-440.
Publisher: SPRINGER VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010.
ISSN: 0171-5216.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 26

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Tumor invasion and metastasis are **complex processes**, requiring the ability of tumor cells to **interact** with **proteins** of the extracellular matrix through **cell-adhesion molecules** on the cell surface. Integrins are heterodimeric membrane glycoproteins, consisting of alpha and beta subunits, which enable cells to recognize adhesive substrates in the

extracellular matrix. The roles of the integrin alpha(5) beta(1) in tumor invasion are highlighted by finding that some tumor cells have lost or reduced alpha(5) beta(1) expression. It therefore functions as a negative signaling regulator. Expression of alpha(5) beta(1) and its mediation of **cell adhesion** in hepatocellular carcinoma (HCC) have not been elucidated. In surgical specimens of HCC we found, by immunohistochemistry and Northern blot analysis, that the alpha(5)-positive rates in cancerous tissues were lower than the corresponding rates in non-cancerous tissues. Reduced expression of the integrin alpha(5) occurred more frequently in HCC with more malignant phenotypes, such as poor differentiation, large size (more than 10-cm in diameter), absence of capsule and high invasion. Reverse transcription/polymerase chain reaction, a more sensitive **assay**, was used to **detect** the alpha(5) mRNA level in LCID20, a highly metastatic model of human HCC, and LCID35, a low-metastasis model. The results showed that integrin alpha(5) was negative in the former and positive in the latter. **Cell adhesion assays** showed the maximal percentage **inhibition** of anti-alpha(5) mAb on SMMC 7721 **cell adhesion** to **fibronectin** to be 68.9 +/- 4.9% at the saturation concentrations of each antibody (200 mu g/ml). If anti-alpha(5) mAb was combined with anti-beta(1) mAb, the **inhibition** was 74.1 +/- 11.1%. It is concluded that reduced expression of the integrin alpha(5) subunit is correlated with more malignant phenotypes of human HCC. Any change in the adhesion of hepatocellular carcinoma cells to **fibronectin** is mainly dependent upon the function of the integrin alpha(5) beta(1).

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ACCESSION NUMBER: 97:536284 SCISEARCH
THE GENUINE ARTICLE: XK146
TITLE: Concerted action of tenascin-C domains in cell adhesion, anti-adhesion and promotion of neurite outgrowth
AUTHOR: Fischer D; BrownLudi M; Schulthess T; ChiquetEhrismann R (Reprint)
CORPORATE SOURCE: FRIEDRICH MIESCHER INST, POB 2543, CH-4002 BASEL, SWITZERLAND (Reprint); FRIEDRICH MIESCHER INST, CH-4002 BASEL, SWITZERLAND; UNIV BASEL, BIOCTR, CH-4056 BASEL, SWITZERLAND
COUNTRY OF AUTHOR: SWITZERLAND
SOURCE: JOURNAL OF CELL SCIENCE, (**JUL 1997**) Vol. 110, Part 13, pp. 1513-1522.
Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE, CAMBS, ENGLAND CB4 4DL.
ISSN: 0021-9533.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 54

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We used a new approach to **identify** domains of chicken tenascin-C required for **interaction** with cells. Instead of expressing the parts of interest, we deleted them from an otherwise intact tenascin-C **molecule** and scored for the concomitant change in activity. As a starting point for all mutant constructs we expressed the smallest naturally occurring tenascin-C splice variant in vertebrate cells. The tenascin-C mutants had either deletions of all EGF-like repeats, all **fibronectin** type III repeats or of the fibrinogen globe. In double mutants the **fibronectin** type III repeats were deleted together with either the EGF-like repeats or the fibrinogen globe, respectively. All tenascin-C variants assembled correctly to hexameric

molecules of the expected molecular characteristics. Intact tenascin-C and the mutant missing the fibrinogen globe did not promote adhesion of chick embryo fibroblasts, whereas both, the hexamers containing solely the fibrinogen globe or the EGF-like repeats were adhesive substrates and even supported cell spreading. When tenascin-C was added to the medium of fibroblasts plated on **fibronectin**-coated wells, **cell adhesion** was **blocked** by intact tenascin-C, but not by mutants missing the fibrinogen globe. In neurite outgrowth **assays** using dorsal root ganglia, **processes** formed on all substrates except on the mutant missing only the fibrinogen globe, where the ganglia failed to adhere. The mutants missing the **fibronectin** type III repeats allowed more rapid neurite outgrowth than all other tenascin-C variants and the mutant consisting essentially of oligomerized EGF-like repeats was as active a substrate for neurite outgrowth as laminin. From the combined data, it is concluded that the activities of intact tenascin-C cannot be mimicked by investigating domain by domain, but the concerted action of several domains leads to the diverse cellular responses.

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ACCESSION NUMBER: 97:159080 SCISEARCH
THE GENUINE ARTICLE: WH722
TITLE: A defect in cell-to-cell adhesion via integrin-fibronectin interactions in a highly metastatic tumor cell line
AUTHOR: Abe Y (Reprint); Tsutsui T; Mu J; Kosugi A; Yagita H; Sobue K; Niwa O; Fujiwara H; Hamaoka T
CORPORATE SOURCE: OSAKA UNIV, SCH MED, FAC MED, DEPT ONCOL, 2-2 YAMADAOKA, SUITA, OSAKA 565, JAPAN (Reprint); OSAKA UNIV, SCH MED, FAC MED, DEPT NEUROCHEM, SUITA, OSAKA 565, JAPAN; OSAKA UNIV, SCH MED, FAC MED, CTR BIOMED RES, SUITA, OSAKA 565, JAPAN; OSAKA UNIV, SCH ALLIED HLTH SCI, FAC MED, SUITA, OSAKA 565, JAPAN; JUNTENDO UNIV, SCH MED, DEPT IMMUNOL, BUNKYO KU, TOKYO 113, JAPAN; HIROSHIMA UNIV, NUCL MED & BIOL RES INST, MINAMI KU, HIROSHIMA 734, JAPAN
COUNTRY OF AUTHOR: JAPAN
SOURCE: JAPANESE JOURNAL OF CANCER RESEARCH, (**JAN 1997**) Vol. 88, No. 1, pp. 64-71.
Publisher: JAPANESE CANCER ASSOCIATION, EDITORIAL OFFICE 7TH FLOOR, JOHKOH BLDG 2-23-11, KOISHIKAWA, TOKYO 112, JAPAN.
ISSN: 0910-5050.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 26

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We investigated the role of integrin-**fibronectin** (FN) **interactions** in tumor **cell adhesion**, Two cloned tumor cell lines designated OV-LM (low-metastatic) and OV-HM (high-metastatic) were isolated from a murine ovarian carcinoma, OV2944. OV-LM and OV-HM cells exhibited high and low RGDS-sequence-dependent adhesiveness to FN, respectively, Both lines expressed comparable levels of alpha 5 and alpha v integrins, which are capable of reacting with RGDS on FN, To compare the functions of these integrins between the two tumor lines, the signaling mechanism following FN stimulation was examined, Significant levels of phosphorylation of focal adhesion kinase (FAK) were **detected** in both OV-LM and OV-HM cells before FN stimulation, Whereas the level of FAK phosphorylation was appreciably enhanced in OV-LM cells stimulated with FN, stimulation of OV-HM cells with FN induced a reduction in the FAK phosphorylation in **association** with a significant **decrease** in the amount of FAK **protein** in

the soluble compartment of cell lysates, A difference in the deposition of FN on the cell surface was also observed between the two types of tumor lines; OV-HM cells had an appreciably smaller amount of FN than OV-LM, Consistent with the functional abnormality of the integrin-FAK system and the smaller amount of FN on OV-HM, this clone exhibited a reduced cell-**cell adhesion** in the in vitro cell aggregation **assay**, Namely, OV-LM cells displayed a time-dependent increase in the formation of cell aggregates, whereas most OV-HM cells remained single, The formation of aggregates by OV-LM cells was **inhibited** by addition of RGDS peptide, These results indicate that the highly metastatic clone, OV-HM, exhibits a **decreased** capacity of cell-**cell adhesion** mediated by integrin-FN **interactions** and suggest that this defect is mainly due to the dysfunction of integrins/FAK rather than a **decrease** in the amount of integrins expressed on tumor cells.

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ACCESSION NUMBER: 96:890064 SCISEARCH
THE GENUINE ARTICLE: VV152
TITLE: Phorbol ester stimulation increases sickle erythrocyte adherence to endothelium: A novel pathway involving alpha(4)beta(1) integrin receptors on sickle reticulocytes and fibronectin
AUTHOR: Kumar A; Eckmam J R; Swerlick R A; Wick T M (Reprint)
CORPORATE SOURCE: GEORGIA INST TECHNOL, SCH CHEM ENGN, 778 ATLANTIC DR, ATLANTA, GA 30332 (Reprint); GEORGIA INST TECHNOL, SCH CHEM ENGN, ATLANTA, GA 30332; EMORY UNIV, SCH MED, DEPT MED, DIV HEMATOL ONCOL, ATLANTA, GA 30322; EMORY UNIV, SCH MED, DEPT DERMATOL, ATLANTA, GA 30322; GEORGIA COMPREHENS SICKLE CELL CTR, ATLANTA, GA
COUNTRY OF AUTHOR: USA
SOURCE: BLOOD, (**1 DEC 1996**) Vol. 88, No. 11, pp. 4348-4358.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399.
ISSN: 0006-4971.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 90

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Sickle-cell adherence to endothelium has been hypothesized to initiate or contribute to microvascular occlusion and pain episodes. Adherence involves plasma **proteins**, endothelial-**cell adhesion molecules**, and receptors on sickle erythrocytes. It has previously been reported that sickle reticulocytes express the alpha(4) beta(1) integrin receptor and **bind** to cytokine-activated endothelium via an alpha(4) beta(1)/vascular-**cell adhesion molecule**-1 (VCAM-1) **interaction**. To elucidate other roles for alpha(4) beta(1) in sickle-cell adherence, the ability of activated alpha(4) beta(1) to promote adhesion to endothelium via a ligand different than VCAM-1 was explored. Adherence **assays** were performed under dynamic conditions at a shear stress of 1 dyne/cm(2). Preincubation of sickle erythrocytes with phorbol 12,13-dibutyrate (PDBu) increased adherence of sickle cells eightfold as compared with untreated sickle cells. Normal erythrocytes, whether treated with PDBu or not, did not adhere to the endothelium. Activating anti-beta(1) antibodies 4B4 and 8A2 also increased the adhesion of sickle, but not normal, red blood cell (RBC) adhesion to endothelium. Anti-alpha(4) antibodies HP1/2 and HP2/1, **inhibitory** antibody 4B5, or an RGD peptide **inhibited** sickle-cell adherence

induced by PDBu. Additional studies were undertaken to examine if **fibronectin**, a ligand for activated alpha(4) beta(1), was involved in PDBu-induced sickle erythrocyte adherence. Adherence of PDBu-treated sickle cells was completely kinhibited by the CS-1 peptide of **fibronectin**. **Fibronectin** was **detected** on the surface of washed endothelium using an antifibronectin antibody in enzyme-linked immunosorbent **assays**. Antifibronectin antibody pretreatment of endothelial cells **inhibited** PDBu-induced adherence by 79% +/- 17%. Incubation of sickle RBCs with exogenous **fibronectin** after PDBu treatment **inhibited** adherence 86% +/- 8%. Taken together, these data suggest that endothelial-bound **fibronectin** mediates adherence of PDBu-treated sickle cells. Interleukin-8 (IL-8), a chemokine released in response to bacterial infection, viral infection, or other injurious agents, and known to activate integrins, also increased adherence of sickle erythrocytes to endothelial cells via **fibronectin**. This novel adherence pathway involving sickle-cell alpha(4) beta(1) activated by PDBu or IL-8 may therefore be relevant in vivo at vascular sites that produce IL-8 or similar agonists in response to vascular injury or immune activation. These observations describe ways in which inflammation and immune responses cause vasoocclusive complications in sickle-cell disease. (C) 1996 by The American Society of Hematology.

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ACCESSION NUMBER: 96:716181 SCISEARCH
THE GENUINE ARTICLE: VJ837
TITLE: DECORIN REGULATES COLLAGENASE GENE-EXPRESSION IN FIBROBLASTS ADHERING TO VITRONECTIN
AUTHOR: HUTTENLOCHER A; WERB Z; TREMBLE P; HUHTALA P; ROSENBERG L; DAMSKY C H (Reprint)
CORPORATE SOURCE: UNIV CALIF SAN FRANCISCO, DEPT STOMATOL, HSW 604, SAN FRANCISCO, CA, 94143 (Reprint); UNIV CALIF SAN FRANCISCO, DEPT ANAT, SAN FRANCISCO, CA, 94143; UNIV CALIF SAN FRANCISCO, RADIOBIOL & ENVIRONM HLTH LAB, SAN FRANCISCO, CA, 94143; MONTEFIORE MED CTR, ORTHOPED RES LABS, BRONX, NY, 10467
COUNTRY OF AUTHOR: USA
SOURCE: MATRIX BIOLOGY, (**SEP 1996**) Vol. 15, No. 4, pp. 239-250.
ISSN: 0945-053X.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 35

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Vitronectin, a principal **cell adhesion molecule** in plasma and extracellular matrix, mediates **cell adhesion** and spreading via the alpha V family of integrins. In this study we demonstrate that decorin, a small dermatan sulfate proteoglycan, regulates extracellular matrix remodeling in rabbit synovial fibroblasts adhering to vitronectin. Decorin induced the expression of the matrix metalloproteinase collagenase (MMP-1) when present on the substrate with vitronectin, or with the 120-kDa cell-**binding** domain of **fibronectin**, but not when present with intact **fibronectin** or Type I collagen. Secreted collagenase was **detected** within 8 h of adhesion; there was no **associated** alteration in cell shape or focal contact formation in cells adhering to decorin plus vitronectin, whereas cell rounding was observed in cells adhering to decorin plus the 120-kDa fragment of **fibronectin**. The core **protein** of decorin, but not the glycosaminoglycan moiety, was sufficient to induce collagenase expression on both substrates; however, the glycosaminoglycan

moiety of decorin as well as the core were required for cell rounding observed in cells adhering to the 120-kDa domain of **fibronectin**. The collagenase-inducing effect of decorin seems to be independent of its effects on transforming growth factor-beta, as function-**blocking** antibodies against transforming growth factor-p did not-interfere with the collagenase-inducing effects of decorin. These data indicate that decorin has specific gene regulatory effects in cells when present in the matrix with vitronectin or the 120-kDa fragment of **fibronectin**, **polypeptides** that are present in actively remodeling tissues. Thus, in combination, these adhesion regulatory **molecules** transduce novel signals that may contribute to the tissue remodeling **process** in morphogenesis, wound healing and disease states.

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ACCESSION NUMBER: 96:511498 SCISEARCH
THE GENUINE ARTICLE: UV225
TITLE: CHANNEL CATFISH, ICTALURUS-PUNCTATUS RAFINESQUE, NEUTROPHIL ADHESION TO SELECTED EXTRACELLULAR-MATRIX PROTEINS, LIPOPOLYSACCHARIDE, AND CATFISH SERUM
AUTHOR: AINSWORTH A J (Reprint); YE Q; XUE L Q; HEBERT P
CORPORATE SOURCE: POB 9825, MISSISSIPPI STATE, MS, 39762 (Reprint); MISSISSIPPI STATE UNIV, COLL VET MED, MISSISSIPPI STATE, MS, 39762
COUNTRY OF AUTHOR: USA
SOURCE: DEVELOPMENTAL AND COMPARATIVE IMMUNOLOGY, (**MAR/APR 1996**) Vol. 20, No. 2, pp. 105-114. ISSN: 0145-305X.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; AGRI
LANGUAGE: ENGLISH
REFERENCE COUNT: 37

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Adhesion of leukocytes to endothelium and extracellular matrix **proteins** is an important step in the inflammatory **process**. Therefore, the adhesion of channel catfish neutrophils to a surface coated with extracellular matrix **proteins**, LPS, and non-immune catfish serum was **evaluated**. Stimulation of neutrophils with phorbol dibutyrate (PDBU) resulted in at least two-fold increases in **cellular adhesion** to all substrates tested except laminin. When EDTA was included during or after PDBU PDBU stimulation, neutrophil adhesion to ECM fibrinogen and LPS coated surfaces was EDTA reduced to the level of unstimulated LPS neutrophils or to 50-60% of that for stimulated neutrophils. Similarly, EDTA and Ca2+/Mg2+ deficient medium reduced homotypic aggregation of PDBU stimulated neutrophils to background levels. Adhesion of stimulated neutrophils to fibrinogen coated surfaces was **inhibited** 44, 33, and 50% when soluble fibrinogen, **fibronectin**, and serum, respectively, were used to **block** the adhesion **assay**. The tripeptide integrin adhesion recognition sequence, Arg-Gly-Asp (RGD), caused 83% reduction and the fibrinogen-**binding inhibitor protein** caused 10% reduction in **binding** of stimulated neutrophils to fibrinogen coated surfaces, Two hexapeptides tested did not reduce neutrophil adhesion to fibrinogen. The **binding** of channel catfish neutrophils to the matrices used in the present study is suggestive that integrin mediated adhesion occurs during biological and pathological **processes** of teleosts. Copyright (C) 1996 Elsevier Science Ltd.

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ACCESSION NUMBER: 96:497812 SCISEARCH
THE GENUINE ARTICLE: UU359

TITLE: HUMAN OSTEOCLAST-LIKE CELLS SELECTIVELY RECOGNIZE LAMININ ISOFORMS, AN EVENT THAT INDUCES MIGRATION AND ACTIVATES CA2+ MEDIATED SIGNALS

AUTHOR: COLUCCI S; GIANNELLI G; GRANO M; FACCIO R; QUARANTA V; ZALLONE A Z (Reprint)

CORPORATE SOURCE: UNIV BARI, INST HUMAN ANAT, @ BARI, ITALY (Reprint); UNIV BARI, INST HUMAN ANAT, @ BARI, ITALY; UNIV BARI, IST CLIN MED 2, @ BARI, ITALY; SCRIPPS CLIN & RES INST, DEPT CELL BIOL, LA JOLLA, CA, 00000

COUNTRY OF AUTHOR: ITALY; USA

SOURCE: JOURNAL OF CELL SCIENCE, (**JUN 1996**) Vol. 109, Part 6, pp. 1527-1535.
ISSN: 0021-9533.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: ENGLISH

REFERENCE COUNT: 53

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Osteoclast precursors are chemotactically attracted to sites of bone resorption via migration pathways that include transendothelial crossing in blood capillaries. Transendothelial migration involves poorly understood **interactions** with basal lamina **molecules**, including laminins. To investigate osteoclast-laminin **interactions**, we used human osteoclast-like cell lines obtained from giant cell tumors of bone (GCT 23 and GCT 24). These cell lines are a well-characterized model for osteoclast functions, such as bone resorption and the behaviour of osteoclast precursors. Both GCT cell lines adhered to laminin-2 (merosin) coated wells in standard adhesion **assays**, but failed to adhere to laminin-1 (EHS-laminin). By light microscopy, GCT cells on laminin-2 were partially spread, with a motile morphology. None of the anti-integrin antibodies tested **inhibited** GCT **cells adhesion** to laminin-2. Peptides containing the integrin adhesion site RGD or the laminin adhesion sequence IKVAV did not **inhibit** GCT **cell adhesion** to laminin-2. By immunofluorescence, beta(1) integrins were organized in focal adhesions. However, in the presence of monensin this reorganization of beta(1) integrins was abolished, indicating that it was probably due to secretion of **fibronectin** by GCT cells subsequent to adhesion to laminin-2. GCT cells transmigrated through membranes coated with laminin-2, much more efficiently than through membranes coated with collagen. Migration was induced by osteocalcin, as a chemoattractant, in a dose-dependent manner. At low osteocalcin concentrations, transmigration was **detectable** on laminin-2 but not collagen. In cells loaded with fura-2, a sharp increase in intracellular Ca2+ was **detected** upon addition of soluble laminin-2, but not laminin-1, due to release from thapsigargin-dependent intracellular stores. In summary, osteoclasts may recognize laminin isoforms differentially. Initial adhesion to laminin-2 appears to be due to integrin-independent mechanisms. Such adhesion, though, may trigger secretion of **fibronectin** that could then support spreading and efficient chemotactic migration. These mechanisms may play an important role in facilitating chemotactic migration of osteoclast precursors toward the bone surface.

L6 ANSWER 21 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 96:372075 SCISEARCH

THE GENUINE ARTICLE: UK557

TITLE: HEMOPHILIC ADHESION MEDIATED BY THE NEURAL CELL-ADHESION MOLECULE INVOLVES MULTIPLE IMMUNOGLOBULIN DOMAINS

AUTHOR: RANHEIM T S (Reprint); EDELMAN G M; CUNNINGHAM B A

CORPORATE SOURCE: SCRIPPS CLIN & RES INST, DEPT NEUROBIOL, 1066 N TORREY PINES RD, LA JOLLA, CA, 92037 (Reprint)

COUNTRY OF AUTHOR: USA
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (**30 APR 1996**) Vol. 93, No. 9, pp. 4071-4075.
ISSN: 0027-8424.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 35
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The neural **cell adhesion molecule** (N-CAM) mediates hemophilic **binding** between a variety of cell types including neurons, neurons and glia, and neurons and muscle cells. The mechanism by which N-CAM on one cell **interacts** with N-CAM on another, however, is unknown. Attempts to **identify** which of the five immunoglobulin-like domains (Ig I-V) and the two **fibronectin** type III repeats (Fn(III) 1-2) in the extracellular region of N-CAM are involved in this **process** have led to ambiguous results. We have generated soluble recombinant **proteins** corresponding to each of the individual immunoglobulin domains and the combined Fn(III) 1-2 and prepared polyclonal antibodies specific for each. The purified **proteins** and antibodies were used in aggregation experiments with fluorescent microspheres and chicken embryo brain cells to determine possible contributions of each domain to homophilic adhesion. The recombinant domains were tested for their ability to **bind** to purified native N-CAM, to **bind** to each other, and to **inhibit** the aggregation of N-CAM on microspheres and the aggregation of neuronal cells. Each of the immunoglobulin domains bound to N-CAM, and in solution all of the immunoglobulin domains **inhibited** the aggregation of N-CAM-coated microspheres. Soluble Ig II, Ig III, and Ig IV **inhibited** neuronal aggregation; antibodies against whole NCAM, the Ig III domain, and the Ig I domain all strongly **inhibited** neuronal aggregation, as well as the aggregation of N-CAM-coated microspheres. Of all the domains, the third immunoglobulin domain alone demonstrated the ability to self-aggregate, whereas Ig I bound to Ig V and Ig II bound to Ig IV. The combined Fn(III) 1-2 exhibited a slight ability to self-aggregate but did not **bind** to any of the immunoglobulinlike domains. These results suggest that N-CAM-N-CAM **binding** involves all five immunoglobulin domains and prompt the hypothesis that in homophilic cell-cell **binding** mediated by N-CAM these domains may **interact** pairwise in an antiparallel orientation.

L6 ANSWER 22 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 95:519132 SCISEARCH
THE GENUINE ARTICLE: RM642
TITLE: ASPARTATE-698 WITHIN A NOVEL CATION-BINDING MOTIF IN ALPHA(4) INTEGRIN IS REQUIRED FOR CELL-ADHESION
AUTHOR: MA L; CONRAD P J; WEBB D L; BLUE M L (Reprint)
CORPORATE SOURCE: BAYER RES CTR, INST BONE & JOINT DISORDERS & CANC, W HAVEN, CT, 06516 (Reprint); BAYER RES CTR, INST BONE & JOINT DISORDERS & CANC, W HAVEN, CT, 06516
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (**04 AUG 1995**) Vol. 270, No. 31, pp. 18401-18407.
ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The **interactions** of alpha(4) beta(1) integrin with vascular **cell adhesion molecule** (VCAM) and **fibronectin** play important roles in many physiological and pathological **processes**, To understand the mechanism of alpha(1) beta(1) integrin-mediated **cell adhesion**, we made mutant alpha(4) constructs, Three aspartic acid (Asp) residues in alpha(4), Asp 489, Asp-698, and Asp-811, were replaced with glutamic acids (Glu), The wild-type and mutant alpha(4) constructs were transfected into K562 cells, and stable transfectants with similar levels of alpha(4) surface expression were established, The Asp --> Glu substitutions did not affect alpha(4) beta(1) **association** or heterodimer formation as demonstrated by immunoprecipitation analysis. However, the glutamate substitutions at Asp-489 and Asp-698 severely impaired **cell adhesion** to VCAM and **fibronectin**, whereas the substitution at Asp-811 had no **detectable** effect on **cell adhesion**. In contrast to these results, isolated alpha(4) beta(1), containing the D489E or D698E substitution, was able to **bind** to VCAM, suggesting that these two residues are not critical for ligand recognition. In searching for a mechanism to explain **inhibition** of adhesion by Asp-489 and Asp 698 mutations, we found that the sequences flanking Asp 698 resemble the DxxxxxD-S-Sx divalent cation/ligand **binding** motif in beta integrins and the I-domains of alpha integrins. This suggests that Asp-698 in the alpha(4) integrin, which does not possess an I-domain, may also be involved in cation **binding** and may be part of a sequence functionally similar to that found in the I-domains of other alpha integrins.

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ACCESSION NUMBER: 95:489158 SCISEARCH
THE GENUINE ARTICLE: BD31P
TITLE: MECHANISMS OF VCAM-1 AND FIBRONECTIN-BINDING TO INTEGRIN ALPHA(4)BETA(1) - IMPLICATIONS FOR INTEGRIN FUNCTION AND RATIONAL DRUG DESIGN
AUTHOR: HUMPHRIES M J (Reprint); SHERIDAN J; MOULD A P; NEWHAM P
CORPORATE SOURCE: UNIV MANCHESTER, SCH BIOL SCI, 2205 STOPFORD BLDG, OXFORD RD, MANCHESTER M13 9PT, LANCS, ENGLAND (Reprint)
COUNTRY OF AUTHOR: ENGLAND
SOURCE: CIBA FOUNDATION SYMPOSIA, (**1995**) Vol. 189, pp. 177-194.
ISSN: 0300-5208.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 43

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Integrin alpha(4) beta(1) can mediate both cell-cell and cell-extracellular matrix adhesion by **binding** to either **fibronectin** or vascular **cell adhesion molecule** 1 (VCAM-1). Both **interactions** are important for extravasation of leukocytes from the blood implying that rationally designed **inhibitors** of alpha(4) beta(1) function may be useful for treating various inflammatory conditions. The mechanisms of ligand **binding** by alpha(4) beta(1) are complicated by the fact that alternative splicing can generate different isoforms of the receptor-**binding** domains in both **fibronectin** and VCAM-1. Therefore, in addition to developing alpha(4) beta(1) antagonists, we have also been interested in **identifying** isoform-specific functions. Recombinant ligand variants have been tested in adhesion and direct receptor-**binding assays** and each **molecule** was found to have a different inherent affinity for alpha(4) beta(1) that endows them with different adhesive activities. This suggests that

alternative splicing may regulate alpha(4) beta(1)-dependent motility in vivo. The initial strategy that we have adopted to develop alpha(4) beta(1) **inhibitors** has been to **identify** key amino acid residues and peptide sequences participating in the receptor-ligand **binding** event and to use this information to generate synthetic mimetics. Three active sites have been identified in **fibronectin** by testing truncated **proteins**, expressing recombinant fragments and screening synthetic peptides. Two of these sites employ versions of a novel integrin-**binding** motif, LDVP/IDAP. A key active site in VCAM-1 has been identified by similar approaches as the related sequence IDSP. Since IDSP-like sequences are probably used by other integrin-**binding** immunoglobulins, derivatives of these peptides may turn out to be the forerunners of a new generation of therapeutic agents with multiple applications.

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ACCESSION NUMBER: 95:482595 SCISEARCH
THE GENUINE ARTICLE: RH640
TITLE: COMPARISON OF THE ANTI-LAMININ ANTIBODY-RESPONSE IN PATIENTS WITH SYSTEMIC LUPUS-ERYTHEMATOSUS (SLE) AND PARASITIC DISEASES (FILARIASIS)
AUTHOR: LERMA J G G (Reprint); MONEO I; DELANDAZURI M O; NAVARRO J S
CORPORATE SOURCE: INST SALUD CARLOS 3, CTR NACL INVEST CLIN & MED PREVENT, DEPT IMMUNOL, E-28029 MADRID, SPAIN (Reprint); HOSP PRINCESA, DEPT IMMUNOL, E-28006 MADRID, SPAIN
COUNTRY OF AUTHOR: SPAIN
SOURCE: CLINICAL IMMUNOLOGY AND IMMUNOPATHOLOGY, (**JUL 1995**) Vol. 76, No. 1, pp. 19-31.
ISSN: 0090-1229.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 46

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB In this study, we analyzed the presence of antibodies against the basement membrane antigen laminin (LMN) in patients with systemic lupus erythematosus (SLE), filariasis, and normal controls. By ELISA, 13.8% of SLE (12/87), 66.7% of parasitized patients (20/30), and two of the normal controls had these antibodies. IgG1 anti-LMN response was elevated in all groups, whereas IgG2 and IgG3 were also elevated in parasitized patients. The analysis of the IgG anti-laminin **binding** capacity in SLE and parasitized patients showed similar average antibody affinity. These antibodies did not react with **fibronectin** by a competition ELISA. By Western blot, the anti-laminin antibodies could be demonstrated in parasitized patient sera but not in SLE sera. Moreover, the ability of these antibodies to **bind** to heat-treated LMN (100 degrees C for 4 min) was different. The study of the **binding** capacity with native or denatured LMN by Western blot and dot-blot **assays** showed that the anti-LMN antibodies from parasitized patients were able to react with both native and denatured forms of LMN, whereas in SLE patients these antibodies were demonstrated only with native LMN. On the other hand, the reactivity **detected** in the normal control sera seems to be different from the anti-LMN antibodies from SLE and parasitized patients, and probably reflects the existence of natural antibodies in these sera. The presence of anti-LMN antibodies correlates significantly with the ability of **inhibition** of U937 **cell adhesion** to LMN-coated surfaces ($P < 0.0025$). The difference of anti-laminin reactivity suggests that antibodies produced following immunization with autoantigens or similar **molecules** present in parasites have different specificities from those spontaneously produced

by individuals with autoimmune diseases. (C) 1995 Academic Press, Inc.

L6 ANSWER 25 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 95:460292 SCISEARCH
THE GENUINE ARTICLE: RF828
TITLE: ALPHA-3A-BETA-1 INTEGRIN LOCALIZES TO FOCAL CONTACTS IN RESPONSE TO DIVERSE EXTRACELLULAR-MATRIX PROTEINS
AUTHOR: DIPERSIO C M; SHAH S; HYNES R O (Reprint)
CORPORATE SOURCE: MIT, HOWARD HUGHES MED INST, CTR CANC RES, CAMBRIDGE, MA, 02139 (Reprint); MIT, HOWARD HUGHES MED INST, CTR CANC RES, CAMBRIDGE, MA, 02139; MIT, DEPT BIOL, CAMBRIDGE, MA, 02139
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL SCIENCE, (**JUN 1995**) Vol. 108, Part 6, pp. 2321-2336.
ISSN: 0021-9533.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 70
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB In vitro **binding assays** and **inhibition** of **cell adhesion** with monoclonal antibodies have implicated the integrin alpha 3 beta 1 as a receptor for a variety of extracellular ligands. However, reports of alpha 3 beta 1-ligand **interactions** are inconsistent, and transfection studies have suggested that alpha 3 beta 1 is not sufficient for cell attachment to ligands other than kalinin/laminin 5. We used immunofluorescence to study subcellular localization of the alpha 3A cytoplasmic domain variant in different cultured cell types. Using standard fixation and permeabilization methods, antibodies specific for alpha 3A stained most cell types in a diffuse pattern, consistent with previous reports. Surprisingly, however, chemical cross-linking of integrins to the extracellular matrix and extraction of the cytoskeleton prior to immunofluorescence revealed alpha 3A in focal contacts of most cells tested, suggesting that the cytoplasmic domain was concealed in intact focal contacts by cytoskeletal or other cytoplasmic **proteins**. The alpha 3A subunit localized to focal contacts in several cell types cultured on **fibronectin**, kalinin/laminin 5, EHS-laminin/laminin 1, type IV collagen, or vitronectin. In contrast, alpha 5 and alpha V integrins were **detected** in focal contacts only in cells grown on their known ligands (**fibronectin**, and **fibronectin** or vitronectin, respectively). Therefore, our results show that alpha 3A beta 1 responds to a broad spectrum of extracellular ligands. Time course comparisons of the recruitment of alpha subunits from different **fibronectin** receptors suggested that localization of alpha 3A beta 1 to **fibronectin**-induced focal contacts was independent of the recruitment of alpha 5 and alpha 4 integrins. However, other studies have shown that alpha 3A beta 1 does not mediate initial **cell adhesion** to many of the ligands that induced its focal contact localization, including **fibronectin**. Therefore, we suggest that alpha 3A beta 1 may be a secondary receptor with post-**cell-adhesion** functions for a broad spectrum of extracellular matrices.

L6 ANSWER 26 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 95:236528 SCISEARCH
THE GENUINE ARTICLE: QM770
TITLE: IMMUNOCHEMICAL ANALYSES OF HUMAN PLASMA FIBRONECTIN-CYTOSOLIC TRANSGLUTAMINASE INTERACTIONS
AUTHOR: ACHYUTHAN K E (Reprint); GOODELL R J; KENNEDYE J R; LEE K

N; HENLEY A; STIEFER J R; BIRCKBICHLER P J
CORPORATE SOURCE: OKLAHOMA MED RES FDN, NOBLE CTR BIOMED RES, 825 NE 13TH ST, OKLAHOMA CITY, OK, 73104 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF IMMUNOLOGICAL METHODS, (**13 MAR 1995**) Vol. 180, No. 1, pp. 69-79. ISSN: 0022-1759.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 33

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB **Fibronectin** is a glycoprotein involved in **cell adhesion**, tissue organization and wound healing. Transglutaminase **binding** and covalent cross-linking of **fibronectin** are physiologically important reactions. We describe microtiter plate-based immunochemical methods to analyze cytosolic transglutaminase-human plasma **fibronectin interactions**. The **method** was sensitive, specific, species-independent and capable of simultaneously analyzing 96 samples for **binding**. **Binding** was time-, temperature- and concentration-dependent and demonstrable with either **protein** immobilized to the plastic. The **assay detected** 1-5 ng transglutaminase or 50 pg **fibronectin** and was comparable in sensitivity to enzyme-linked immunosorbent **assays**. CaCl2 (8 mM) enhanced transglutaminase **binding** by two-fold. Molar concentrations of NaCl or millimolar concentrations of chloride salts of barium, copper or zinc **inhibited binding** by 50-60%. The **binding** was also competitively **blocked** by soluble **fibronectin** (IC50 = 2.3 nM) or by anti-**fibronectin** IgG (IC50 = 0.5 mu M). Inclusion of dithiothreitol or 2-mercaptoethanol during **binding** resulted in a concentration-dependent **inhibition** of transglutaminase-**fibronectin interactions** (IC50 = 1.5 mM and 20 mM, respectively). A **complex** of [anti-transglutaminase IgG-transglutaminase-**fibronectin**-anti-**fibronectin** IgG] suggested that the **binding** sites and antibody epitopes could overlap, but are distinct and surface-exposed in the two **proteins**. Liver transglutaminase bound **fibronectin** 30-50% less compared to erythrocyte transglutaminase. **Fibronectin**-transglutaminase affinity was adequate for quantitating either antigen in lysates of lung fibroblasts, breast carcinomas or Escherichia coli. These immunochemical analyses will be useful for determining the affinity and mapping the domains involved in antibody recognition or **protein-protein interactions** using recombinant **molecules** of transglutaminase and **fibronectin**.

L6 ANSWER 27 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 94:717434 SCISEARCH
THE GENUINE ARTICLE: PQ490
TITLE: THE SHORT AMINO-ACID-SEQUENCE PRO-HIS-SER-ARG-ASN IN HUMAN FIBRONECTIN ENHANCES CELL-ADHESIVE FUNCTION
AUTHOR: AOTA S; NOMIZU M; YAMADA K M (Reprint)
CORPORATE SOURCE: NIDR, DEV BIOL LAB, BLDG 30, RM 421, BETHESDA, MD, 20892 (Reprint); NIDR, DEV BIOL LAB, BETHESDA, MD, 20892
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (**07 OCT 1994**) Vol. 269, No. 40, pp. 24756-24761. ISSN: 0021-9258.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH

REFERENCE COUNT: 22

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Synergistic sites in the central cell-adhesive domain of **fibronectin** (FN) substantially enhance **cell adhesion** mediated by the alpha(5) beta(1) integrin receptor for **fibronectin**. We characterized a critical minimal sequence needed for synergistic activity using site directed mutagenesis and homology scanning using intramolecular chimeras. The minimal cell-**binding** domain of FN consisting of the 9th and 10th type III FN repeat was expressed in an Escherichia coli expression system. This **protein** retained high biological activity when **assayed** using a competitive **inhibition assay** for FN-mediated adhesion of baby hamster kidney or HT-1080 cells. In contrast, a construct consisting of the 8th and 10th repeat displayed very low biological activity. By replacing various portions of the 8th repeat with homologous 9th repeat segments, we mapped the synergistic region to the center of the 9th repeat. When a very short peptide sequence, Pro-His-Ser-Arg-Asn (PHSRN), from the 9th repeat was substituted for the homologous pentapeptide site in the 8th repeat sequence, the recombinant **protein** showed markedly enhanced activity. Further mutagenesis analysis suggested that the arginine residue of this pentapeptide sequence is important for function. We also identified a weaker adjacent synergy region other than the PHSRN region, Epitope mapping of an anti-FN monoclonal antibody that **inhibits** FN-mediated adhesion identified the same critical regions. A synthetic peptide containing the PHSRN sequence showed neither competitive **inhibitory** activity in solution nor synergy with a soluble RGD-containing peptide. However, when the same synthetic peptide was positioned via a covalent bond at the corresponding site of the normally inactive 8th repeat, it mediated an enhancement of adhesive activity. These results **identify** a pentapeptide site that synergistically enhances the cell-adhesive activity of the FN RGD sequence.

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ACCESSION NUMBER: 94:702695 SCISEARCH

THE GENUINE ARTICLE: PP694

TITLE: ICAM-3 REGULATES LYMPHOCYTE MORPHOLOGY AND INTEGRIN-MEDIATED T-CELL INTERACTION WITH ENDOTHELIAL-CELL AND EXTRACELLULAR-MATRIX LIGANDS

AUTHOR: CAMPANERO M R; SANCHEZMATEOS P; DELPOZO M A; SANCHEZMADRID F (Reprint)

CORPORATE SOURCE: UNIV AUTONOMA MADRID, HOSP PRINCESA, SERV IMMUNOL, C-DIEGO LEON 62, E-28006 MADRID, SPAIN (Reprint); UNIV AUTONOMA MADRID, HOSP PRINCESA, SERV IMMUNOL, E-28006 MADRID, SPAIN

COUNTRY OF AUTHOR: SPAIN

SOURCE: JOURNAL OF CELL BIOLOGY, (**NOV 1994**) Vol. 127, No. 3, pp. 867-878.
ISSN: 0021-9525.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: ENGLISH

REFERENCE COUNT: 60

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Leukocyte activation is a **complex process** that involves multiple cross-regulated **cell adhesion** events. In this report, we investigated the role of intercellular adhesion **molecule**-3 (ICAM-3), the third identified ligand for the beta 2 integrin leukocyte function-**associated** antigen-1 (LFA-1), in the regulation of leukocyte adhesion to ICAM-1, vascular **cell adhesion molecule**-1 (VCAM-1), and the 38- and 80-kD fragments of **fibronectin** (FN40 and FN80). The activating

anti-ICAM-3 HP2/19, but not other anti-ICAM-3 mAb, was able to enhance T lymphoblast adhesion to these **proteins** when combined with very low doses of anti-CD3 mAb, which were unable by themselves to induce this phenomenon. In contrast, anti-ICAM-1 mAb did not enhance T cell attachment to these substrata. T **cell adhesion** to ICAM-1, VCAM-1, FN40, and FN80 was specifically **blocked** by anti-LFA-1, anti-VLA alpha 4, and anti-VLA alpha 5 mAb, respectively. The activating anti-ICAM-3 HP2/19 was also able to specifically enhance the VLA-4- and VLA-5-mediated **binding** of leukemic T Jurkat cells to VCAM-1, FN40, and FN80, even in the absence of cooccupancy of the CD3-TcR **complex**. We also studied the localization of ICAM-3, LFA-1, and the VLA beta 1 integrin, by immunofluorescence microscopy, on cells **interacting** with ICAM-1, VCAM-1 and FN80. We found that the anti-ICAM-3 HP2/19 mAb specifically promoted a dramatic change on the morphology of T lymphoblasts when these cells were allowed to **interact** with those adhesion ligands. Under these conditions, it was observed that a large cell contact area from which an uropod-like structure (heading uropod) was projected toward the outer milieu. However, when T blasts were stimulated with other adhesion promoting agents as the activating anti-VLA beta 1 TS2/16 mAb or phorbol esters, this structure was not **detected**. The anti-ICAM-3 TP1/24 mAb was also unable to induce this phenomenon. Notably, a striking cell redistribution of ICAM-3 was induced specifically by the HP2/19 mAb, but not by the other anti-ICAM-3 mAb or the other adhesion promoting agents. Thus, ICAM-3 was almost exclusively concentrated in the most distal portion of the heading uropod whereas either LFA-1 or the VLA beta 1 integrin were uniformly distributed all over the large contact area. Moreover, this phenomenon was also observed when T cells were specifically stimulated with the HP2/19 mAb to **interact** with TNF alpha-activated endothelial cells. We found the localization of linear arrays of myosin within the heading uropod. In contrast, actin-based cytoskeleton presented a uniform distribution over the broad contact area with the substrate. In addition, butanedione monoxime, a myosin-disrupting drug, abolished both the morphological cell change and ICAM-3 clustering. Altogether, these results demonstrate that ICAM-3 has a regulatory role on multiple pathways of T **cell adhesion** and morphology.

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ACCESSION NUMBER: 94:559299 SCISEARCH
THE GENUINE ARTICLE: PF176
TITLE: SPECIFIC ROLES OF THE ALPHA-V-BETA-1, ALPHA-V-BETA-3 AND ALPHA-V-BETA-5 INTEGRINS IN AVIAN NEURAL CREST CELL-ADHESION AND MIGRATION ON VITRONECTIN
AUTHOR: DELANNET M; MARTIN F; BOSSY B; CHERESH D A; REICHARDT L F; DUBAND J L (Reprint)
CORPORATE SOURCE: UNIV PARIS 07, INST JACQUES MONOD, BIOL CELLULAIRE DEV LAB, 2 PL JUSSIEU, F-75251 PARIS 05, FRANCE (Reprint); UNIV PARIS 07, INST JACQUES MONOD, BIOL CELLULAIRE DEV LAB, F-75251 PARIS 05, FRANCE; UNIV CALIF SAN FRANCISCO, HOWARD HUGHES MED INST, SAN FRANCISCO, CA, 94143; SCRIPPS CLIN & RES INST, DEPT IMMUNOL, LA JOLLA, CA, 92037
COUNTRY OF AUTHOR: FRANCE; USA
SOURCE: DEVELOPMENT, (**SEP 1994**) Vol. 120, No. 9, pp. 2687-2702.
ISSN: 0950-1991.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 72

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB To **identify** potentially important extracellular matrix

adhesive **molecules** in neural crest cell migration, the possible role of vitronectin and its corresponding integrin receptors was examined in the adhesion and migration of avian neural crest cells in vitro. Adhesion and migration on vitronectin were comparable to those found on **fibronectin** and could be almost entirely abolished by antibodies against vitronectin and by RGD peptides. Immunoprecipitation and immunocytochemistry analyses revealed that neural crest cells expressed primarily the alpha V beta 1, alpha V beta 3 and alpha V beta 5 integrins as possible vitronectin receptors. **Inhibition assays** of **cellular adhesion** and migration with function-perturbing antibodies demonstrated that adhesion of neural crest cells to vitronectin was mediated essentially by one or more of the different alpha V integrins, with a possible preeminence of alpha V beta 1, whereas cell migration involved mostly the alpha V beta 3 and alpha V beta 5 integrins. Immunofluorescence labeling of cultured motile neural crest cells revealed the alpha V integrins are differentially distributed on the cell surface. The beta 1 and alpha V subunits were both diffuse on the surface of cells and in focal adhesion sites in **association** with vinculin, talin and alpha-actinin, whereas the alpha V beta 3 and alpha V beta 5 integrins were essentially diffuse on the cell surface. Finally, vitronectin could be **detected** by immunoblotting and immunohistochemistry in the early embryo during the ontogeny of the neural crest. It was in particular closely **associated** with the surface of migrating neural crest cells. In conclusion, our study indicates that neural crest cells can adhere to and migrate on vitronectin in vitro by an RGD-dependent mechanism involving at least the alpha V beta 1, alpha V beta 3 and alpha V beta 5 integrins and that these integrins may have specific roles in the control of **cell adhesion** and migration.

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ACCESSION NUMBER: 94:260670 SCISEARCH
THE GENUINE ARTICLE: NJ950
TITLE: NEURONAL CHONDROITIN SULFATE PROTEOGLYCAN NEUROCAN BINDS TO THE NEURAL CELL-ADHESION MOLECULES NG-CAM/L1/NILE AND N-CAM, AND INHIBITS NEURONAL ADHESION AND NEURITE OUTGROWTH
AUTHOR: FRIEDLANDER D R (Reprint); MILEV P; KARTHIKEYAN L; MARGOLIS R K; MARGOLIS R U; GRUMET M
CORPORATE SOURCE: NYU, MED CTR, DEPT PHARMACOL, 550 1ST AVE, NEW YORK, NY, 10016 (Reprint); NYU, MED CTR, DEPT NEUROL, NEW YORK, NY, 10016; SUNY HLTH SCI CTR, DEPT PHARMACOL, BROOKLYN, NY, 11203
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL BIOLOGY, (**MAY 1994**) Vol. 125, No. 3, pp. 669-680.
ISSN: 0021-9525.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 59

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We have previously shown that aggregation of microbeads coated with N-CAM and Ng-CAM is **inhibited** by incubation with soluble neurocan, a chondroitin sulfate proteoglycan of brain, suggesting that neurocan **binds** to these **cell adhesion molecules** (Grumet, M., A. Flaccus, and R. U. Margolis. 1993. J. Cell Biol. 120:815). To investigate these **interactions** more directly, we have tested **binding** of soluble I-125- neurocan to microwells coated with different glycoproteins. Neurocan bound at high levels to Ng-CAM and N-CAM, but little or no **binding** was

detected to myelin-**associated** glycoprotein, EGF receptor, **fibronectin**, laminin, and collagen IV. The **binding** to Ng-CAM and N-CAM was saturable and in each case Scatchard plots indicated a high affinity **binding** site with a dissociation constant of similar to 1 nM. **Binding** was significantly reduced after treatment of neurocan with chondroitinase, and free chondroitin sulfate **inhibited binding** of neurocan to Ng-CAM and N-CAM. These results indicate a role for chondroitin sulfate in this **process**, although the core glycoprotein also has **binding** activity. The COOH-terminal half of neurocan was shown to have **binding** properties essentially identical to those of the full-length proteoglycan.

To study the potential biological functions of neurocan, its effects on neuronal adhesion and neurite growth were analyzed. When neurons were incubated on dishes coated with different combinations of neurocan and Ng-CAM, neuronal adhesion and neurite extension were inhibited. Experiments using anti-Ng-CAM antibodies as a substrate also indicate that neurocan has a direct inhibitory effect on neuronal adhesion and neurite growth. Immunoperoxidase staining of tissue sections showed that neurocan, Ng-CAM, and N-CAM are all present at highest concentration in the molecular layer and fiber tracts of developing cerebellum. The overlapping localization in vivo, the molecular binding studies, and the striking effects on neuronal adhesion and neurite growth support the view that neurocan may modulate neuronal adhesion and neurite growth during development by binding to neural cell adhesion molecules.

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ACCESSION NUMBER: 94:226083 SCISEARCH
THE GENUINE ARTICLE: NB502
TITLE: EXTRACELLULAR-MATRIX ACCUMULATION IN IMMUNE-MEDIATED TUBULOINTERSTITIAL INJURY
AUTHOR: TANG W W; FENG L L; XIA Y Y; WILSON C B (Reprint)
CORPORATE SOURCE: SCRIPPS CLIN & RES INST, DEPT IMMUNOL IMM5, 10666 N TORREY PINES RD, LA JOLLA, CA, 92037 (Reprint); SCRIPPS CLIN & RES INST, DEPT IMMUNOL IMM5, LA JOLLA, CA, 92037
COUNTRY OF AUTHOR: USA
SOURCE: KIDNEY INTERNATIONAL, (**APR 1994**) Vol. 45, No. 4, pp. 1077-1084.
ISSN: 0085-2538.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 40

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The accumulation of excessive extracellular matrix (ECM) following tubular injury likely represents an imbalance between ECM production and degradation. We assessed the temporal relationship between the accumulation of ECM, **cell adhesion molecules**, matrix degrading **proteinases**, and their **inhibitors** in a rat model of anti-tubular basement membrane (TBM) antibody-**associated** tubulointerstitial nephritis (TIN) by the RNase protection **assay** and immunohistochemistry. There was an increase in the steady state expression of **fibronectin** (FN) and alpha(2)(IV) collagen mRNAs beginning on day 7 with the onset of neutrophil infiltration. An increase in alpha(1)(III) collagen and alpha(1)-integrin did not occur until days 9 and 10, respectively, at which time mononuclear leukocytes were the predominant infiltrating cell. Increased levels of FN, alpha(1)(III), alpha(2)(IV) and alpha(1)-integrin mRNAs occurred through day 14. By immunohistochemistry, increased accumulation of collagen IV, heparan sulfate proteoglycan, and laminin were **detected** along the thickened TBM; collagens I and III were

immunolocalized within the tubulointerstitium, while FN was present in both the TBM and interstitium in rats with TIN on day 14. The increase in matrix accumulation was **associated** with little or no increase in **proteinases**. u-PA transcripts fell beginning on day 8, with recovery to control values by day 12. Transin mRNA was found at low levels only on days 8 and 9, and the **protein** could not be **detected** by Western blotting. In contrast, these changes were **associated** with an increase in **proteinase inhibitors**, so that TIMP and PAI-1 mRNAs increased beginning on day 7 and persisted through day 14. PAI-1 mRNA correlated with biologic activity, while TIMP was immunolocalized within the peritubular endothelium and infiltrating leukocytes. These data demonstrate a temporal **association** between ECM accumulation, a minimal change in **proteinase**, and an increase in **proteinase inhibitors**.

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ACCESSION NUMBER: 94:192809 SCISEARCH
THE GENUINE ARTICLE: NC592
TITLE: ADULT ADENOHYPOPHYSEAL CELLS EXPRESS BETA(1) INTEGRINS AND PREFER LAMININ DURING CELL-SUBSTRATUM ADHESION
AUTHOR: HORACEK M J (Reprint); KAWAGUCHI T; TERRACIO L
CORPORATE SOURCE: CREIGHTON UNIV, SCH PHARM & ALLIED HLTH PROFESS, DEPT PHYS THERAPY, 2500 CALIF PLAZA, OMAHA, NE, 68178 (Reprint); UNIV S CAROLINA, SCH MED, DEPT DEV BIOL & ANAT, COLUMBIA, SC, 29208
COUNTRY OF AUTHOR: USA
SOURCE: IN VITRO CELLULAR & DEVELOPMENTAL BIOLOGY-ANIMAL, (**JAN 1994**) Vol. 30A, No. 1, pp. 35-40.
ISSN: 0883-8364.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 54

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Beta1 Integrins are a family of structurally related heterodimeric cell surface receptors that are involved in adhesion to **molecules** in the extracellular matrix (ECM) such as laminin (LN), **fibronectin** (FN), and collagen. These receptors are expressed by many cell types and mediate a variety of **processes** such as cell-matrix and cell-to-**cell adhesion**, cell migration, growth, and differentiation. The purpose of these studies was to **identify** and partially characterize beta1 integrins on adenohypophyseal cells and to begin to elucidate their functional importance. Adenohypophyses were removed from adult male rats, dispersed using 0.25% trypsin, rinsed, and resuspended in a 1:1 mixture of Dulbecco's modified Eagle's medium and F12 medium containing 10% fetal bovine serum and antibiotics. Ten million cells were allowed to attach to each of five plastic culture dishes overnight. The next day, the adenohypophyseal cells were surface-labeled with I-125. The labeled cells were lysed and centrifuged. The supernatant was immunoprecipitated using preimmune IgGs (100 mug/ml) and was then incubated with a polyclonal antibody against the rat beta1 family of integrins or with a variety of immune IgGs directed against the a subunit of the receptor (anti alpha1 anti alpha2, anti alpha3, and anti alpha5 antibodies). The receptors were then immunoprecipitated by addition of **protein** A-Sepharose or IgG1 Sepharose. After washing, the immunoprecipitates were subjected to sodium dodecyl sulfate polyacrylamide gel electrophoresis and autoradiography. Cultured adenohypophyseal cells expressed the beta1 integrin subunit, which was **associated** with the alpha1 alpha2, alpha3, and alpha5 integrin subunits. These integrins are known to have **binding** specificities for LN, FN, epiligrin,

and several collagens. Immunocytochemical staining and confocal microscopy verified that these receptors were present on the cell surface in vitro. The addition of anti rat beta1 integrin antibodies to dispersed adenohypophyseal cells partially **blocked** their attachment to ECM ligands in **cell adhesion assays**. In addition, peptides containing Agr-Gly-Asp-Ser (RGDS) partially **blocked** adenohypophyseal cell attachment to FN and to a lesser extent to LN. These studies show for the first time that adult adenohypophyseal cells express several beta1 integrin dimers and attach to ECM ligands corresponding to their **binding** specificities. The fact that these **interactions** are only partially **blocked** by RGDS peptides and antibodies against the beta1 family of integrins may indicate that other cell-matrix receptors are also present. Additional studies are necessary to determine whether these **interactions** have a functional significance (such as an effect on hormone secretion) beyond their role in cell-matrix adhesion.

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ACCESSION NUMBER: 93:662800 SCISEARCH
THE GENUINE ARTICLE: MD800
TITLE: DECREASED ADHESION TO ENDOTHELIAL-CELLS AND MATRIX PROTEINS OF H-2KB GENE TRANSFECTED TUMOR-CELLS
AUTHOR: LAURI D; DEGIOVANNI C; BIONDELLI T; LALLI E; LANDUZZI L; FACCHINI A; NICOLETTI G; NANNI P; DEJANA E; LOLLINI P L (Reprint)
CORPORATE SOURCE: UNIV BOLOGNA, IST CANCEROL, VIALE FILOPANTI 22, I-40126 BOLOGNA, ITALY; IST RIC FARMACOL MARIO NEGRI, MILAN, ITALY; IST, BIOTECHNOL SATELLITE UNIT, BOLOGNA, ITALY; CNR, IST CITOMORFOL, CHIETI, ITALY; IST SCI RIZZOLI, BOLOGNA, ITALY
COUNTRY OF AUTHOR: ITALY
SOURCE: BRITISH JOURNAL OF CANCER, (**NOV 1993**) Vol. 68, No. 5, pp. 862-867.
ISSN: 0007-0920.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 49

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Transfection of murine metastatic B78H1 cells (derived from B16 melanoma) with a syngeneic H-2K(b) gene was used to study the effect of Major Histocompatibility **Complex** (MHC) gene products on tumour **cell adhesion** to endothelial cells and matrix **proteins** and the involvement in the metastatic **process**. H-2K(b)-expressing transfectants showed a reduced adhesion to endothelial surfaces of different origin (four murine endotheliomas and human umbilical vein endothelial cells) when compared to parental B78H1 cells and to controls transfected with pSV2neo alone. On the average a 50-70% reduction in adhesion to endothelial cells was observed among H-2K(b) transfectants. H-2K(b) transfectants had a reduced expression of the alpha4 integrin subunit, moreover the adhesion of Neo-transfected clones to endothelial cells was reduced to the levels of H-2K(b) transfectants by antibodies directed against the beta1 subunit and the endothelial VCAM-1 **molecule**, thus suggesting an impairment of the VLA-4/VCAM-1 **interaction** in H-2K(b) transfectants. Adhesion to extracellular matrix components was also strongly **decreased**: in general the adhesion of H-2K(b) cells showed a 50-75% **inhibition** with respect to Neo or parental controls. The highest difference was observed in adhesion to vitronectin and laminin, the lowest in adhesion to **fibronectin**. Reduction in adhesive properties of H-2K(b)-expressing transfectants could be involved in the reduced

metastatic ability, **evaluated** by means of intravenous injection of cells: H-2K(b) transfectants yielded less than ten lung colonies, while all controls produced more than 100. Our data indicate that expression of a single class I MHC gene can significantly alter the metastatic phenotype of MHC-negative tumour cells and this could be related to a general alteration of tumour cell adhesive **interactions**.

L6 ANSWER 34 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 93:606594 SCISEARCH
THE GENUINE ARTICLE: LZ829
TITLE: INTERACTIONS OF PROMONOCYTIC U937 CELLS WITH PROTEINS OF THE EXTRACELLULAR-MATRIX
AUTHOR: PUCILLO C E M; COLOMBATTI A (Reprint); VITALE M; SALZANO S; ROSSI G; FORMISANO S
CORPORATE SOURCE: CTR RIFERIMENTO ONCOL, DIV ONCOL SPERIMENTALE 2, VIA PEDEMONTANA OCCIDENTALE 12, I-33081 AVIANO, ITALY; UNIV UDINE, DIPARTIMENTO SCI & TECNOL BIOMED, I-33100 UDINE, ITALY; UNIV NAPLES, DIPARTIMENTO BIOL & PATOL MOLEC & CELLULAIRE, I-80138 NAPLES, ITALY
COUNTRY OF AUTHOR: ITALY
SOURCE: IMMUNOLOGY, (**OCT 1993**) Vol. 80, No. 2, pp. 248-252.
ISSN: 0019-2805.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 27
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Monocyte **interaction** with **proteins** of the extracellular matrix (ECM) is regulated by expression of specific cell-surface receptors. 12-O-tetradecanoyl phorbol-13-acetate (TPA) has been shown to induce the promonocytic cell line U937 to a more differentiated monocyte-like state. In this study we have analysed the attachment of U937 cells to ECM **proteins** and the effects of treatment with TPA on this **process**. Non-induced U937 cells attach to **fibronectin**- and Matrigel-coated surfaces without TPA stimulation, but TPA further increases adherence to these substrates as measured by an enhanced **binding** and by the lower concentration of **proteins** needed in the substrate to achieve 50% of maximal **cell adhesion**. Attachment to type I collagen was seen only with activated U937 cells, whereas no measurable attachment to bovine serum albumin, vitronectin, and type IV collagen was **detected**. TPA-activated U937 cells showed a two-fold increase in the expression of the RGD-dependent integrin receptors alpha3 and alpha5, and a reduction in the expression of alpha4, another **fibronectin**-specific receptor, whereas the common beta1 chain was unchanged. Attachment of U937 cells to **fibronectin** was primarily mediated by the alpha3 and alpha5 integrins, as revealed by the ability of GRGDS peptides to **inhibit** attachment, whereas the CS-1 peptide, containing the alpha4 **binding** site, was largely ineffective in **blocking** attachment.

L6 ANSWER 35 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 92:703145 SCISEARCH
THE GENUINE ARTICLE: KA675
TITLE: TYROSINE PHOSPHORYLATION OF MEMBRANE-PROTEINS MEDIATES CELLULAR INVASION BY TRANSFORMED-CELLS
AUTHOR: MUELLER S C; YEH Y Y; CHEN W T (Reprint)
CORPORATE SOURCE: GEORGETOWN UNIV, SCH MED, DEPT ANAT & CELL BIOL, 3900 RESERVOIR RD NW, WASHINGTON, DC, 20007

COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL BIOLOGY, (**DEC 1992**) Vol. 119, No. 5, pp. 1309-1325.
ISSN: 0021-9525.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 48

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Tyrosine phosphorylation of membrane-**associated proteins** is involved at two distinct sites of contact between cells and the extracellular matrix: adhesion plaques (**cell adhesion** and de-adhesion) and invadopodia (invasion into the extracellular matrix). Adhesion plaques from chicken embryonic fibroblasts or from cells transformed by Rous sarcoma virus contain low levels of tyrosine-phosphorylated **proteins** (YPPs) which were below the level of **detection** in 0.5-mum thin, frozen sections. In contrast, intense localization of YPPs was observed at invadopodia of transformed cells at sites of degradation and invasion into the **fibronectin**-coated gelatin substratum, but not in membrane extensions free of contact with the extracellular matrix. Local extracellular matrix degradation and formation of invadopodia were **blocked** by genistein, an **inhibitor** of tyrosine-specific kinases, but cells remained attached to the substratum and retained their free-membrane extensions. Invadopodia reduced or lost YPP labeling after treatment of the cells with genistein, but adhesion plaques retained YPP labeling. The plasma membrane contact fractions of normal and transformed cells have been isolated form cells grown on gelatin cross-linked substratum using a novel fractionation scheme, and analyzed by immunoblotting. Four major YPPs (150, 130, 81, and 77 kD) characterize invadopodial membranes in contact- with the matrix, and are probably responsible for the intense YPP labeling **associated** with invadopodia extending into sites of matrix degradation. YPP150 may be an invadopodial-specific YPP since it is approximately 3.6-fold enriched in the invasive contact fraction relative to the cell body fraction and is not observed in normal contacts. YPP130 is enriched in transformed cell contacts but may also be present in normal contacts. The two major YPPs of normal contacts (130 and 71 kD) are much lower in abundance than the major tyrosine-phosphorylated bands **associated** with invadopodial membranes, and likely represent major adhesion plaque YPPs. YPP150, paxillin, and tensin appear to be enriched in the cell contact fractions containing adhesion plaques and invadopodia relative to the cell body fraction, but are also present in the soluble supernate fraction. However, vinculin, talin, and alpha-actinin that are localized at invadopodia, are equally concentrated in cell bodies and cell contacts as is the membrane-adhesion receptor beta1 integrin. Thus, tyrosine phosphorylation of the membrane-bound **proteins** may contribute to the cytoskeletal and plasma membrane events leading to the formation and function of invadopodia that contact and proteolytically degrade the extracellular matrix; we have identified several candidate YPPs that may participate in the regulation of these **processes**.

L6 ANSWER 36 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 92:485517 SCISEARCH
THE GENUINE ARTICLE: JJ092
TITLE: ALPHA-4/BETA-1 INTEGRIN (VLA-4) LIGANDS IN ARTHRITIS - VASCULAR CELL-ADHESION MOLECULE-1 EXPRESSION IN SYNOVIUM AND ON FIBROBLAST-LIKE SYNOVIOCYTES
AUTHOR: MORALESDUCRET J; WAYNER E; ELICES M J; ALVAROGRACIA J M; ZVAIFLER N J; FIRESTEIN G S (Reprint)
CORPORATE SOURCE: UNIV CALIF SAN DIEGO, MED CTR, DIV RHEUMATOL 8417, 225

DICKINSON ST, SAN DIEGO, CA, 92103; UNIV MINNESOTA, MINNEAPOLIS, MN, 55455; CYTEL CORP, SAN DIEGO, CA, 92121
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF IMMUNOLOGY, (**15 AUG 1992**) Vol. 149, No. 4, pp. 1424-1431.
ISSN: 0022-1767.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 43

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Expression of vascular **cell adhesion molecule**-1 (VCAM-1) in synovial tissue was determined using the immunoperoxidase technique. Normal, rheumatoid arthritis (RA), and osteoarthritis (OA) synovia bound VCAM-1 antibodies in the intimal lining as well as blood vessels. The amount of VCAM-1 was significantly greater in the synovial lining of RA and OA tissues compared with normal synovium ($p < 0.002$). There was also a trend toward greater levels of VCAM-1 staining in blood vessels of arthritic tissue (RA > OA > normal). Because VCAM-1 staining was especially intense in the synovial lining, VCAM-1 expression and regulation was studied on cultured fibroblast-like synoviocytes (FLS) derived from this region. Both VCAM-1 and intercellular adhesion **molecule** 1 were constitutively expressed on FLS. VCAM-1 expression was further increased by exposure to IL-1-beta, TNF-alpha, IL-4, and IFN-gamma. These cytokines (except for IL-4) also induced intercellular adhesion **molecule** 1 expression on FLS. ELAM was not **detected** on resting or cytokine-stimulated FLS. The specificity of VCAM-1 for FLS was demonstrated by the fact that only trace amounts were **detected** on normal and RA dermal fibroblasts. Cytokines induced intercellular adhesion **molecule** 1 display on dermal fibroblasts but had minimal effect on VCAM-1 expression. Finally, in adherence **assays**, Jurkat cell **binding** to resting FLS monolayers was **inhibited** by antibody to alpha-4/beta-1 integrin (VLA-4), CS-1 peptide from alternatively spliced **fibronectin** (which is another VLA-4 ligand), and, to a lesser extent, anti-VCAM-1 antibody. After cytokine stimulation of FLS, Jurkat-**binding** significantly increased, and this increase was **blocked** by anti-VCAM-1 antibody. Therefore, both CS-1 and VCAM-1 participate in VLA-4-mediated adherence to resting FLS in vitro, and VCAM-1 is responsible for the increase in Jurkat **binding** mediated by cytokines.

L6 ANSWER 37 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 92:422359 SCISEARCH
THE GENUINE ARTICLE: JC512
TITLE: A CELL-SURFACE HEPARAN-SULFATE PROTEOGLYCAN MEDIATES NEURAL CELL-ADHESION AND SPREADING ON A DEFINED SEQUENCE FROM THE C-TERMINAL CELL AND HEPARIN BINDING DOMAIN OF FIBRONECTIN, FN-C/H-II
AUTHOR: HAUGEN P K (Reprint); LETOURNEAU P C; DRAKE S L; FURCHT L T; MCCARTHY J B
CORPORATE SOURCE: UNIV MINNESOTA, DEPT CELL BIOL & ANAT, MINNEAPOLIS, MN, 55455; UNIV MINNESOTA, DEPT LAB MED & PATHOL, MINNEAPOLIS, MN, 55455; UNIV MINNESOTA, CTR BIOMED ENGN, MINNEAPOLIS, MN, 55455
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF NEUROSCIENCE, (**JUL 1992**) Vol. 12, No. 7, pp. 2597-2608.
ISSN: 0270-6474.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE

LANGUAGE: ENGLISH
REFERENCE COUNT: 84

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB FN-C/H II is a heparin **binding** synthetic peptide from the C-terminal cell and heparin **binding** domain of **fibronectin** (FN) that mediates neuronal **cell adhesion**, spreading, and neurite outgrowth. Cellular **interactions** with FN-C/H II are **inhibited** by soluble heparin, suggesting that a cell-surface proteoglycan may mediate **interactions** with FN-C/H II (Haugen et al., 1990). To test this hypothesis further, heparan sulfate (HS) or chondroitin sulfate (CS) was removed from the cell surface by enzyme treatment. Heparitinase but not chondroitinase treatment of cells **inhibited** rat B104 neuroblastoma **cell adhesion** and spreading on FN-C/H II. Additionally, heparitinase treatment **decreased** the spreading of cells on the 33/66 kDa fragments containing the C-terminal heparin **binding** domain of FN. Furthermore, antibodies generated against a mouse melanoma HS proteoglycan (HSPG) **inhibited** B104 **cell adhesion** to FN-C/H II and the 33/66 kDa FN fragments. S-35-HSPG isolated from B104 cells directly bound to FN-C/H II both in solid phase **assays** and by affinity chromatography, but failed to **bind** to a control peptide from this region, CS1. The **binding** of S-35-HSPG was predominantly mediated by the HS and not the core **protein** of the HSPG. SDS-PAGE of iodinated HSPG demonstrated a single 78 kDa core **protein** following heparitinase digestion, which migrated at 51 kDa under nonreducing conditions. Anti-HSPG antibodies recognized the 78 kDa core **protein** by immunoblotting, and stained the surface of rat B104 neuroblastoma cells and cells of the primary neonatal rat nervous system. These results **identify** a cell-surface HSPG that likely mediates neuronal cell **binding interactions** with FN-C/H II.

L6 ANSWER 38 OF 67 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 91:594284 SCISEARCH
THE GENUINE ARTICLE: GM039
TITLE: PHAGOCYTIC CELL MOLECULES THAT BIND THE COLLAGEN-LIKE REGION OF C1Q - INVOLVEMENT IN THE C1Q-MEDIATED ENHANCEMENT OF PHAGOCYTOSIS
AUTHOR: GUAN E; BURGESS W H; ROBINSON S L; GOODMAN E B; MCTIGUE K J; TENNER A J (Reprint)
CORPORATE SOURCE: AMER RED CROSS, BIOCHEM LAB, BIOMED RES & DEV, 15601 CRABBS BRANCH WAY, ROCKVILLE, MD, 20855; AMER RED CROSS, MOLEC BIOL LAB, BIOMED RES & DEV, BETHESDA, MD, 20855
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (**1991**) Vol. 266, No. 30, pp. 20345-20355.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 82

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB C1q **binds** to and elicits cellular responses by several cell types, including monocytes, macrophages, neutrophils, B cells, and fibroblasts. The cell-**binding** domain is located within the collagen-like pepsin-resistant region of the C1q **molecule** (C1q tails). An affinity matrix of C1q tails coupled to Sepharose was used to select C1q-**binding proteins** from detergent extracts of surface-iodinated human monocytes, polymorphonuclear leukocytes, and the U937 cells. The major radiolabeled **polypeptide** eluted specifically from the ligand affinity column had an apparent molecular mass (M(r)) of 126,000. Minor iodinated components eluted from

Sepharose-tails migrated with M(r) of 216,000 and 55,000. When subjected to sodium dodecyl sulfate-polyacrylamide gel electrophoresis under reducing conditions no change in the migration of any of these **polypeptide** bands was **detected**. None of these **polypeptides** reacted with antibodies directed against the integrins alpha-5-beta-1 (**fibronectin** receptor) or alpha-upsilon-beta-3 (vitronectin receptor), LFA-1, or to several other **cell adhesion molecules**. The M(r) 126,000 band was found to contain more than one **polypeptide**. Lectin **binding** properties, susceptibility to glycosidases and proteases, and immunoreactivity with the monoclonal antibody L-10, indicated that CD43 (sialophorin/leukosialin) is a component of this band. However, further data show that a monoclonal antibody, generated by immunization with the isolated C1q-**binding** fractions, recognizes a cell surface sialoglycoprotein distinct from CD43 and **inhibits** the C1q-mediated enhancement of phagocytosis in monocytes. These latter observations provide the first definitive connection between a specific phagocytic cell surface **protein** and a known C1q-mediated function. While these **proteins** contain sialic acid, **binding assays** and functional **assays** using neuraminidase-treated cells demonstrate that the functional **interaction** between C1q and the cell surface is not via sialic acid. The data taken together indicate either that the functional C1q receptor on phagocytic cells is a multisubunit **complex** or that multiple **proteins** can **interact** with the fragment of C1q containing the cell-**binding** domain, at least one of which is involved in the C1q-mediated enhancement of phagocytosis.

L6 ANSWER 39 OF 67 USPATFULL on STN

ACCESSION NUMBER:	2005:26380 USPATFULL			
TITLE:	Peptides with β1 integrin subunit dependent cell adhesion modulating activity			
INVENTOR(S):	McCarthy, James B., Minneapolis, MN, United States Furcht, Leo T., Minneapolis, MN, United States Frey, Angela Brienzo, Waukesha, WI, United States			
PATENT ASSIGNEE(S):	Regents of the University of Minnesota, Minneapolis, MN, United States (U.S. corporation)			

	NUMBER	KIND	DATE		
PATENT INFORMATION:	US 6849712	B1	20050201		
	WO 9937669		19990729		<--
APPLICATION INFO.:	US 2000-600432		20001002	(9)	
	WO 1999-US1236		19990121		
			20001002	PCT 371 date	

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1998-96212P	19980812	(60)
	US 1998-96211P	19980812	(60)
	US 1998-72119P	19980122	(60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	GRANTED
PRIMARY EXAMINER:	Weber, Jon P.
ASSISTANT EXAMINER:	Lukton, David
LEGAL REPRESENTATIVE:	Mueting Raasch & Gebhardt, P.A.
NUMBER OF CLAIMS:	27
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	19 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT:	1174

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Peptides capable of modulating β1 integrin subunit dependent cell

adhesion which includes a C-terminal aromatic amino acid residue and an amino acid residue having a lipophilic alkyl side chain as the penultimate C-terminal residue are provided. These "LipAr" C-terminated peptides are typically capable of modulating the β1 integrin subunit dependent adhesion of cells, such as Ramos cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 40 OF 67 USPATFULL on STN

ACCESSION NUMBER:	2004:26863 USPATFULL			
TITLE:	Inhibitors of α4β1 mediated cell adhesion			
INVENTOR(S):	Blinn, James, Lawton, MI, United States Chrusciel, Robert, Portage, MI, United States Fisher, Jed, Kalamazoo, MI, United States Tanis, Steven, Kalamazoo, MI, United States Thomas, Edward, Kalamazoo, MI, United States Lobl, Thomas, Foster City, CA, United States Teegarden, Bradley, San Diego, CA, United States			
PATENT ASSIGNEE(S):	Pharmacia & Upjohn Company, Kalamazoo, MI, United States (U.S. corporation) Tanabe Seiyaku Co., Ltd., Osaka, JAPAN (non-U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6685617	B1	20040203	
	WO 9967230		19991229	<--
APPLICATION INFO.:	US 2001-720088		20010309	(9)
	WO 1999-US14233		19990623	
	NUMBER		DATE	
PRIORITY INFORMATION:	US 1998-90421P		19980623	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	GRANTED			
PRIMARY EXAMINER:	Raymond, Richard L.			
LEGAL REPRESENTATIVE:	Browdy and Neimark, P.L.L.C.			
NUMBER OF CLAIMS:	9			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	0 Drawing Figure(s); 0 Drawing Page(s)			
LINE COUNT:	7144			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to compound of formula (I), that are potent inhibitors of α.sub.4β.sub.1 mediated adhesion to either VCAM or CS-1 and which could be useful for the treatment of inflammatory diseases. Specifically, the molecules of the present invention can be used for treating or preventing α.sub.4β.sub.1 adhesion mediated conditions in a mammal such as a human. This method may comprise administering to a mammal or a human patient an effective amount of the compound or composition as explained in the present specification. ##STR1##

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 41 OF 67 USPATFULL on STN

ACCESSION NUMBER:	2000:124769 USPATFULL
TITLE:	Epiligrin, an epithelial ligand for integrins
INVENTOR(S):	Carter, William G., Bainbridge Island, WA, United States Gil, Susana G., Seattle, WA, United States Ryan, Maureen C., Bellevue, WA, United States
PATENT ASSIGNEE(S):	Fred Hutchinson Cancer Research Center, Seattle, WA,

United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6120991		20000919	<--
	WO 9506660		19950309	<--
APPLICATION INFO.:	US 1996-600982		19960222	(8)
	WO 1994-US10261		19940902	
			19960222	PCT 371 date
			19960222	PCT 102(e) date

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-115918, filed on 2 Sep 1993, now abandoned And a continuation-in-part of Ser. No. US 1994-292065, filed on 17 Aug 1994, now abandoned which is a continuation of Ser. No. US 1993-154638, filed on 18 Nov 1993, now abandoned which is a continuation of Ser. No. US 1991-654103, filed on 8 Feb 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-607137, filed on 30 Oct 1990, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Houtteman, Scott W.
LEGAL REPRESENTATIVE: Christensen O'Connor Johnson & Kindness PLLC
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 102 Drawing Figure(s); 68 Drawing Page(s)
LINE COUNT: 4826
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Nucleic acid sequences are disclosed encoding an E170 epithelial ligand and capable of hybridizing under stringent conditions to the nucleotide sequences derived from cDNA clones shown in the figure. Also disclosed are vectors containing the nucleic acid sequences, and cells transformed with the vectors. Methods are given for purifying and utilizing epiligrin, an epithelial glycoprotein complex, and its component glycoproteins, and for raising antibodies against components of this complex. Assay methods are further provided for identification of functional epiligrin in tissues.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 42 OF 67 USPATFULL on STN
ACCESSION NUMBER: 1998:134832 USPATFULL
TITLE: Method for identifying a target peptide that modulates the binding of epinectin ligand to integrin receptors
INVENTOR(S): Carter, William Gene, Winslow, WA, United States
PATENT ASSIGNEE(S): Fred Hutchinson Cancer Research Center, Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5830678		19981103	<--
APPLICATION INFO.:	US 1996-643770		19960506	(8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1994-292065, filed on 17 Aug 1994, now abandoned which is a continuation of Ser. No. US 1993-154638, filed on 18 Nov 1993, now abandoned which is a continuation of Ser. No. US 1991-654103, filed on 8 Feb 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-607137, filed on 30 Oct 1990, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Eisenschenk, Frank C.

ASSISTANT EXAMINER: Nolan, Patrick
LEGAL REPRESENTATIVE: Christensen O'Connor Johnson & Kindness PLLC
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 49 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 2596
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A substantially pure epinectin covalently linked glycoprotein complex is disclosed having a ligand portion binding at least to the α.sub.3 β.sub.1 integrin receptor for use in modifying cellular adhesion to a substratum. Also disclosed are specific binding partners for epinectin, as exemplified by monoclonal antibody, and α.sub.3 β.sub.1 and α.sub.6 β.sub.4 integrin receptor peptides for use as inhibitors, antagonists, and agonists of receptor binding to epinectin ligand.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 43 OF 67 USPATFULL on STN
ACCESSION NUMBER: 96:14791 USPATFULL
TITLE: Polypeptides for promoting cell attachment
INVENTOR(S): Ginsberg, Mark H., San Diego, CA, United States
Plow, Edward F., San Diego, CA, United States
Bowditch, Ronald, Encinitas, CA, United States
PATENT ASSIGNEE(S): The Scripps Research Institute, La Jolla, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5492890		19960220	<--
APPLICATION INFO.:	US 1991-804224		19911205	(7)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1991-803623, filed on 27 Nov 1991 which is a continuation-in-part of Ser. No. US 1991-725600, filed on 3 Jul 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-620668, filed on 3 Dec 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Wax, Robert A.
ASSISTANT EXAMINER: Hendricks, Keith D.
LEGAL REPRESENTATIVE: Fitting, Thomas
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 2351
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel polypeptides derived from human **fibronectin**, and fusion proteins containing those peptide sequences are described which define a receptor binding site on **fibronectin** that binds to the platelet receptor glycoprotein GPIIb-IIIa expressed by cells. The receptor binding site of human **fibronectin** includes at least **fibronectin** amino acid residues 1410-1436. The polypeptides facilitate attachment of cells to substrates either alone or in conjunction with RGD-containing peptides. Vectors preparing the fusion proteins and antibodies are also described. Methods for promoting cell attachment and for **inhibiting cell adhesion** are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 44 OF 67 USPATFULL on STN
ACCESSION NUMBER: 93:104814 USPATFULL

TITLE: Method for diagnosing non-healing ulcers
INVENTOR(S): Grinnell, Frederick, Dallas, TX, United States
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System, Austin, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5270168		19931214	<--
APPLICATION INFO.:	US 1991-795667		19911121	(7)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1990-483207, filed on 21 Feb 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Rosen, Sam
LEGAL REPRESENTATIVE: Arnold, White & Durkee
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 10 Drawing Page(s)
LINE COUNT: 1554

AB The present invention provides methods for the diagnosis of non-healing ulcers in humans. Provided are methods for detecting the presence of non-healing ulcers by assaying for certain cell adhesion-related proteins or their degradation products in ulcer exudate. The methods of the present invention are useful as an initial, quick and inexpensive screening process for a condition which is often misdiagnosed. It has been discovered that in non-healing ulcers there appear to be proteases which degrade cell adhesion-related proteins, e.g., fibronectin and vitronectin. Protein separation techniques, such as electrophoresis, may be used in combination with immunoassay techniques to isolate and identify these degradation products, as well as the cell adhesion-related proteins themselves.

L6 ANSWER 45 OF 67 USPATFULL on STN
ACCESSION NUMBER: 93:5469 USPATFULL
TITLE: Adhesion receptor for laminin and its use
INVENTOR(S): Ruoslahti, Erkki I., Rancho Santa Fe, CA, United States
Engvall, Eva, Rancho Santa Fe, CA, United States
Gehlsen, Kurt R., San Diego, CA, United States
PATENT ASSIGNEE(S): La Jolla Cancer Research Foundation, La Jolla, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5180809		19930119	<--
APPLICATION INFO.:	US 1989-357354		19890525	(7)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1988-196986, filed on 20 May 1988, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Wax, Robert A.
ASSISTANT EXAMINER: Walsh, Stephen
LEGAL REPRESENTATIVE: Pretty Schroeder Brueggemann & Clark
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 849
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB An adhesion receptor for laminin is provided. The receptor is isolated from cell or tissue extracts and fractionated on an affinity column composed of cell attachment-promoting fragments of laminin coupled to Sepharose.TM. in the presence of divalent cations. This receptor can be

used to prepare specific antibodies for the analysis of the amount of laminin receptor expressed by cells and has other applications in cellular and tumor biology.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 46 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 2001079285 PCTFULL ED 20020826
TITLE (ENGLISH): METHODS AND COMPOSITIONS FOR THE TREATMENT OF FIBROTIC CONDITIONS AND IMPAIRED LUNG FUNCTION AND TO ENHANCE LYMPHOCYTE PRODUCTION
TITLE (FRENCH): PROCEDES ET COMPOSITIONS SERVANT A TRAITER DES ETATS FIBREUX ET L'ALTERATION DE LA FONCTION PULMONAIRE, ET A AMELIORER LA PRODUCTION DE LYMPHOCYTES
INVENTOR(S): PILON, Aprile, L.;
WELCH, Richard, W.;
FARROW, Jeffrey;
MELBY, James;
WIESE, Laura;
LOHNAS, Gerald;
MIELE, Lucio;
ANTICO, Giovanni
PATENT ASSIGNEE(S): CLARAGEN, INC.
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2001079285	**A1**	**20011025**

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW GH GM KE LS MW MZ SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE TR BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.:	WO 2001-US12126	A	20010413
PRIORITY INFO.:	2000-09/549,926		20000414
	US 2000-09/549,926		20000414

ABEN The present invention provides methods and compositions to treat fibrotic conditions, to increase lymphocyte production in vivo, and to improve and/or normalize lung function, pulmonary compliance, blood oxygenation, and blood pH to **inhibit** inflammatory processes to stimulate or **inhibit** pro-inflammatory and immune cells, and to **inhibit** migration of vascular endothelial cells. The invention contemplates the administration of human uteroglobin, native or recombinant, as a means of achieving these ends. Specifically, it has been found that uteroglobin **inhibits cell adhesion** to **fibronectin**, increases lymphocyte production in vivo, and improves and/or normalizes lung function, pulmonary compliance, blood oxygenation, and blood pH, and **inhibits** inflammatory process. In addition it has been found that uteroglobin can stimulate or **inhibit** pro-inflammatory and immune cells and **inhibitor** migration of vascular endothelial cells.

ABFR L'invention concerne des procedes et compositions servant a traiter des etats fibreux, a augmenter in vivo la production de lymphocytes et a ameliorer et/ou normaliser la fonction pulmonaire, la compliance pulmonaire, l'oxygenation sanguine et le pH sanguin, de maniere a inhiber des processus inflammatoires afin de stimuler ou inhiber des cellules pro-inflammatoires et immunes, et a inhiber la migration des

cellules endotheliales vasculaires. A cette fin, l'invention consiste a administrer de l'uteroglobine humaine, naturelle ou recombinee. On a notamment trouve que l'uteroglobine inhibait l'adhesion cellulaire a la fibronectine, augmentait la production de lymphocytes in vivo et ameliorait et/ou normalisait la fonction pulmonaire, la compliance pulmonaire, l'oxygenation sanguine et le pH sanguin, et inhibait le processus inflammatoire. En outre, on a trouve que l'uteroglobine pouvait stimuler ou inhiber des cellules pro-inflammatoires et immunes et inhiber la migration de cellules endotheliales vasculaires.

L6 ANSWER 47 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 2001000677 PCTFULL ED 20020828
TITLE (ENGLISH): A METHOD OF MODULATING INTEGRIN MEDIATED CELLULAR ACTIVITY AND AGENTS USEFUL FOR SAME
TITLE (FRENCH): PROCEDE SERVANT A MODULER UNE ACTIVITE CELLULAIRE PROVOQUEE PAR INTEGRINE ET AGENTS DE MODULATION UTILES
INVENTOR(S): AGREZ, Michael, Valentine
PATENT ASSIGNEE(S): THE UNIVERSITY OF NEWCASTLE RESEARCH ASSOCIATES LIMITED;
AGREZ, Michael, Valentine
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2001000677	**A1**	**20010104**

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW MZ SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.:	WO 2000-AU729	A	20000628
PRIORITY INFO.:	1999-PQ 1248		19990628
	AU 1999-PQ 1248		19990628
	AU 2000-PQ 8003		20000606
	AU 2000-PQ 8003		20000606

ABEN There is disclosed agents capable of inhibiting the binding of a MAP kinase to a binding domain of an integrin for the MAP kinase, and methods of modulating the activity of a cell utilising the agents. The methods are particularly suitable for inhibiting the growth of cancer cells.
ABFR

L6 ANSWER 48 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 2000055206 PCTFULL ED 20020515
TITLE (ENGLISH): ENDOTHELIAL CELL STIMULATION BY A COMPLEX OF FIBRONECTIN AND VASCULAR ENDOTHELIAL GROWTH FACTOR
TITLE (FRENCH): STIMULATION DE CELLULES ENDOTHELIALES A L'AIDE D'UN COMPLEXE DE FIBRONECTINE ET DE FACTEUR DE CROISSANCE ENDOTHELIALE VASCULAIRE
INVENTOR(S): WIJELATH, Errol, S.;
MURRAY-WIJELATH, Jacqueline;
HAMMOND, William, P.
PATENT ASSIGNEE(S): THE HOPE HEART INSTITUTE;
WIJELATH, Errol, S.;
MURRAY-WIJELATH, Jacqueline;
HAMMOND, William, P.
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent

PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2000055206	**A1**	**20000921**

DESIGNATED STATES
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW GH GM KE LS MW SD SL SZ TZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 2000-US7183 A 20000317
PRIORITY INFO.: 1999-60/125,006 19990318
US 1999-60/125,006 19990318

ABEN The present invention pertains to isolated complexes containing the growth factor VEGF in association with the adhesion protein fibronectin or fragments thereof, and to methods of administering the complexes i(in vitro) or i(in vivo) to promote or induce endothelial cell migration, angiogenesis and wound healing.

ABFR La presente invention concerne des complexes isoles contenant le facteur de croissance VEGF en association avec la fibronectine de proteine d'adhesion ou de fragments de cette derniere et des procedes d'administration desdits complexes i(in vitro)ou i(in vivo) afin de favoriser ou d'induire la migration des cellules endotheliales, l'angiogenese et la cicatrisation des blessures.

L6 ANSWER 49 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 2000055181 PCTFULL ED 20020515
TITLE (ENGLISH): METHODS OF MODULATING CELL ATTACHMENT AND MIGRATION
TITLE (FRENCH): METHODES PERMETTANT DE MODULER LA FIXATION ET LA MIGRATION DES CELLULES
INVENTOR(S): GOETINCK, Paul
PATENT ASSIGNEE(S): THE GENERAL HOSPITAL CORPORATION
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2000055181	**A1**	**20000921**

DESIGNATED STATES
W: CA JP AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE

APPLICATION INFO.: WO 2000-US6740 A 20000315
PRIORITY INFO.: 1999-60/124,396 19990315
US 1999-60/124,396 19990315

ABEN The invention features a **method** of modulating, e.g., **inhibiting** or promoting, the spatial or positional relationship of a cell to a substrate, or modulating the intracellular response of a cell to a substrate, i(in vitro) or i(in vivo). The **method** includes administering an agent which modulates the **interaction**, e.g., the **binding**, of the syndecan-4 ectodomain with a counterligand, thereby modulating the spatial or positional relationship of a cell to a substrate, or modulating the intracellular response of a cell to a substrate. The preferred

counterligand is an ECM component, e.g., the heparin-**binding** domain of a component of the extracellular matrix (ECM) such as **fibronectin**, vitronectin, a laminin or a collagen. The invention also features methods of **identifying compounds** which modulate, e.g., **inhibit** or promote, the spatial or positional relationship of a cell to a substrate, or modulate the intracellular response of a cell to a substrate, and methods of treating a subject having a disorder characterized by unwanted or abnormal **cell adhesion** or spreading, e.g., cancer.

ABFR L'invention concerne une methode permettant de moduler, c'est-a-dire d'inhiber ou de favoriser, la relation spatiale ou positionnelle d'une cellule vis-a-vis d'un substrat, ou de moduler la reponse intracellulaire d'une cellule a un substrat, in vitro ou in vivo. Ladite methode consiste a administrer un agent modulant l'interaction, c'est-a-dire la liaison, de l'ectodomaine du syndecan-4 avec un contre-ligand, ce qui permet de moduler la relation spatiale ou positionnelle d'une cellule vis-a-vis d'un substrat, ou de moduler la reponse intracellulaire d'une cellule a un substrat. Le contre-ligand prefere est un composant de la matrice extracellulaire, notamment le domaine de liaison de l'heparine d'un composant de la matrice extracellulaire, tel que la fibronectine, la vitronectine, une laminine ou un collagene. L'invention concerne egalement des methodes d'identification de composes qui modulent, c'est-a-dire qui inhibent ou favorisent, la relation spatiale ou positionnelle d'une cellule vis-a-vis d'un substrat, ou qui modulent la reponse intracellulaire d'une cellule a un substrat, ainsi que des methodes de traitement des sujets atteint d'une pathologie caracterisee par une adhesion ou une proliferation cellulaire anormale ou indesirable, comme le cancer.

L6 ANSWER 50 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER:	2000021986 PCTFULL ED 20020515
TITLE (ENGLISH):	MATRIX-REMODELING GENES
TITLE (FRENCH):	GENES DE REMODELAGE DE MATRICE
INVENTOR(S):	WALKER, Michael, G.; VOLKMUTH, Wayne; KLINGLER, Tod, M.
PATENT ASSIGNEE(S):	INCYTE PHARMACEUTICALS, INC.; WALKER, Michael, G.; VOLKMUTH, Wayne; KLINGLER, Tod, M.
LANGUAGE OF PUBL.:	English
DOCUMENT TYPE:	Patent
PATENT INFORMATION:	

NUMBER	KIND	DATE
WO 2000021986	**A2**	**20000420**

DESIGNATED STATES

W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU

ZW GH GM KE LS MW SD SL SZ TZ UG ZW AM AZ BY KG KZ MD
RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC
NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 1999-US23315 A 19991006
PRIORITY INFO.: 1998-09/169,289 19981009
US 1998-09/169,289 19981009

ABEN The invention provides novel matrix-remodeling genes and polypeptides encoded by those genes.
The invention also provides expression vectors, host cells, antibodies, agonists, and antagonists.
The invention also provides methods for diagnosing, treating or preventing diseases associated with
matrix remodeling.

ABFR L'invention porte sur de nouveaux genes de remodelage de matrice et sur des polypeptides codes
par ces genes. L'invention porte egalement sur des vecteurs d'expression, des cellules hotes, des
anticorps, des agonistes et des antagonistes. L'invention porte, de plus, sur des procedes de
diagnostic, de traitement ou de prevention de maladies associees au remodelage de matrice.

L6 ANSWER 51 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 2000002587 PCTFULL ED 20020515
TITLE (ENGLISH): CANCER TREATMENT METHODS USING THERAPEUTIC CONJUGATES THAT BIND TO AMINOPHOSPHOLIPIDS
TITLE (FRENCH): PROCEDES DE TRAITEMENT DU CANCER METTANT EN APPLICATION DES CONJUGUES THERAPEUTIQUES SE FIXANT A DES AMINOPHOSPHOLIPIDES
INVENTOR(S): THORPE, Philip, E.;
RAN, Sophia
PATENT ASSIGNEE(S): BOARD OF REGENTS, THE UNIVERSITY OF TEXAS SYSTEM
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2000002587	**A1**	**20000120**

DESIGNATED STATES
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK
EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP
KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL
PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU
ZA ZW GH GM KE LS MW SD SL SZ UG ZW AM AZ BY KG KZ MD
RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC
NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 1999-US15668 A 19990712
PRIORITY INFO.: 1998-60/092,589 19980713
US 1998-60/092,589 19980713
US 1998-60/110,600 19981202
US 1998-60/110,600 19981202

ABEN Disclosed is the surprising discovery that aminophospholipids, such as phosphatidylserine and
phosphatidylethanolamine, are specific, accessible and stable markers of the luminal surface of
tumor blood vessels. The present invention thus provides aminophospholipid-targeted diagnostic and
therapeutic constructs for use in tumor intervention.
Antibody-therapeutic agent conjugates and
constructs that bind to aminophospholipids are particularly provided, as are methods of specifically
delivering therapeutic agents, including toxins and coagulants, to the

stably-expressed
aminophospholipids of tumor blood vessels, thereby inducing thrombosis, necrosis and tumor
regression.

ABFR On a decouvert que des aminophospholipides, tels que phosphatidylserine et
phosphatidylethanolamine, sont des marqueurs specifiques, accessibles et stables de la surface
intracavitaire de vaisseaux sanguins tumoraux. L'invention concerne, de ce fait, des produits de
recombinaison diagnostiques et therapeutiques ciblant les aminophospholipides et concus pour
intervenir sur la tumeur. Elle concerne en particulier des conjugues d'agents therapeutiques et
d'anticorps et des produits de recombinaison se fixant aux aminophospholipides, ainsi que des
procedes servant a administrer de facon specifique des agents therapeutiques, y compris des toxines
et des coagulants, aux aminophospholipides d'expression stable de vaisseaux sanguins tumoraux, ce
qui provoque une thrombose, une necrose et une regression de la tumeur.

L6 ANSWER 52 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 2000002584 PCTFULL ED 20020515
TITLE (ENGLISH): CANCER TREATMENT METHODS USING ANTIBODIES TO AMINOPHOSPHOLIPIDS
TITLE (FRENCH): PROCEDES DE TRAITEMENT DU CANCER REPOSANT SUR L'UTILISATION D'ANTICORPS VIS-A-VIS DES AMINOPHOSPHOLIPIDES
INVENTOR(S): THORPE, Philip, E.;
RAN, Sophia
PATENT ASSIGNEE(S): BOARD OF REGENTS, THE UNIVERSITY OF TEXAS SYSTEM
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 2000002584	**A2**	**20000120**

DESIGNATED STATES
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZA ZW GH GM KE LS MW SD SL SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.:	WO 1999-US15600	A	19990712
PRIORITY INFO.:	1998-60/092,672		19980713
	US 1998-60/092,672		19980713
	US 1998-60/110,608		19981202
	US 1998-60/110,608		19981202

ABEN Disclosed are the surprising discoveries that aminophospholipids, such as phosphatidylserine
and phosphatidylethanolamine, are stable and specific markers accessible on the luminal surface of
tumor blood vessels, and that the administration of an anti-aminophospholipid antibody alone is
sufficient to induce thrombosis, tumor necrosis and tumor regression i(in vivo). This invention
therefore provides anti-aminophospholipid antibody-based methods and compositions for use in the
specific destruction of tumor blood vessels and in the treatment of

solid tumors. Although various
antibody conjugates and combinations are thus provided, the use of naked, or unconjugated,
anti-phosphatidylserine antibodies is a particularly important aspect of the invention, due to
simplicity and effectiveness of the approach.

ABFR L'invention concerne la decouverte surprenante selon laquelle les aminophospholipides, du type
phosphatidylserine et phosphatidylethanolamine, sont des marqueurs stables et accessibles a la
surface intracavitaire des vaisseaux sanguins de tumeur, et selon laquelle la simple administration
d'anticorps vis-a-vis des aminophospholipides suffit a induire la thrombose, la necrose tumorale et
la regression tumorale i(in vivo). En consequence, l'invention concerne des procedes reposant sur
l'utilisation d'anticorps vis-a-vis des aminophospholipides, et des compositions destinees a etre
utilisees pour la destruction specifique des vaisseaux sanguins de tumeur et le traitement des
tumeurs solides. Bien que l'invention concerne ainsi plusieurs conjugues et combinaisons
d'anticorps, l'utilisation d'anticorps nus ou non conjugues vis-a-vis du type phosphatidylserine est
un aspect particulierement important de l'invention, grace a la simplicite et a l'efficacite de
l'approche considereea

L6 ANSWER 53 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER:	1999064627 PCTFULL ED 20020515
TITLE (ENGLISH):	PROBES USED FOR GENETIC FILING
TITLE (FRENCH):	SONDES UTILISEES POUR PROFILAGE GENETIQUE
INVENTOR(S):	ROBERTS, Gareth, Wyn
PATENT ASSIGNEE(S):	GENOSTIC PHARMA LIMITED; ROBERTS, Gareth, Wyn
LANGUAGE OF PUBL.:	English
DOCUMENT TYPE:	Patent
PATENT INFORMATION:	

NUMBER	KIND	DATE
WO 9964627	**A2**	**19991216**

DESIGNATED STATES

W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW GH GM KE LS MW SD SL SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.:	WO 1999-GB1780	A 19990604
PRIORITY INFO.:	1998-9812099.1	19980606
	GB 1998-9812099.1	19980606
	GB 1998-9813291.3	19980620
	GB 1998-9813291.3	19980620
	GB 1998-9813611.2	19980624
	GB 1998-9813611.2	19980624
	GB 1998-9813835.7	19980627
	GB 1998-9813835.7	19980627
	GB 1998-9814110.4	19980701
	GB 1998-9814110.4	19980701
	GB 1998-9814580.8	19980707

GB 1998-9814580.8	19980707
GB 1998-9815438.8	19980716
GB 1998-9815438.8	19980716
GB 1998-9815576.5	19980718
GB 1998-9815576.5	19980718
GB 1998-9815574.0	19980718
GB 1998-9815574.0	19980718
GB 1998-9816085.6	19980724
GB 1998-9816085.6	19980724
GB 1998-9816086.4	19980724
GB 1998-9816086.4	19980724
GB 1998-9816921.2	19980805
GB 1998-9816921.2	19980805
GB 1998-9817097.0	19980807
GB 1998-9817097.0	19980807
GB 1998-9817200.0	19980808
GB 1998-9817200.0	19980808
GB 1998-9817632.4	19980814
GB 1998-9817632.4	19980814
GB 1998-9817943.5	19980819
GB 1998-9817943.5	19980819

ABEN People vary enormously in their response to disease and also in their response to therapeutic interventions aimed at ameliorating the disease process and progression. However, the provision of medical care and medical management is centered around observations and protocols developed in clinical trials on groups or cohorts of patients. This group data is used to derive a standardised method of treatment which is subsequently applied on an individual basis. There is considerable evidence that a significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiological response. In order to bring about the integration of genomics into medical practice and enable design and building of a technology platform which will enable the everyday practice of molecular medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiological states of interest. According to the invention, the number of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide critical clinical information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clinical prognostic information - 'genostics'. The GenosticTM profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate

healthcare resources to
those deemed most in need. The use of our invention could also lead to a host of new applications
for such profiling technologies, such as identification of persons with particular work or
environment related risk, selection of applicants for employment, training or specific opportunities
or for the enhancing the planning and organisation of health services, education services and social
services.

ABFR La reaction d'un patient a une maladie ou a des interventions therapeutiques ayant pour but
d'ameliorer le processus ou la progression d'une maladie varie enormement. L'administration de soins
medicaux et la surveillance medicale sont donc effectuees a partir d'observations et de protocoles
developpes dans des essais cliniques sur des groupes ou des cohortes de patients. Ces donnees sont
utilisees afin de deduire un procede de traitement standardise, qui est ensuite applique sur une
base individuelle. Il a ete prouve qu'un facteur significatif important dont depend la variabilite
de la reaction individuelle a la maladie, a la therapie, et au pronostic reside dans le constituant
genetique de la personne. De nombreux exemples montrent que les polymorphismes d'un gene donne
peuvent alterer la fonction de la proteine codee par ledit gene, ce qui provoque une reaction
physiologique variable. Dans le but d'integrer la genomique a la pratique medicale, de concevoir et
de construire une plate-forme technologique qui permette la mise en oeuvre quotidienne de la
medecine moleculaire, il est necessaire de mettre sur pied un mode d'alignement des donnees des
sequences d'ADN sur l'identification des genes jouant un role primordial dans l'apparition, le
developpement, la progression et l'issue d'une maladie ou d'etats physiologiques determines. Selon
l'invention, le nombre de genes et leurs configurations (mutations et polymorphismes) qu'il est
indispensable d'identifier, de maniere a obtenir des informations cliniques critiques concernant le
pronostic individuel, est considerablement inferieur aux 100 000 genes censes composer le genome
humain. L'identification du groupe de genes principal permet de mettre sur pied des technologies de
profilage genetique, consistant a identifier le groupe principal et les variants des sequence
indispensables pour obtenir une large base d'informations pronostiques cliniques permettant
l'identification des genes par la genomique. Le profilage genomique TM des patients ou des personnes
ameliorera radicalement la planification, la gestion de l'administration des soins de sante, et le
ciblage des ressources en soins medicaux appropries pour ceux qui en ont le plus besoin, par les
cliniciens, les professionnels de la sante et autres parties. L'invention permet egalement d'obtenir
un nombre important de nouvelles applications de ces technologies de profilage, telles que
l'identification des personnes en fonction du risque d'un travail particulier ou d'un environnement,

la selection des candidats pour des postes de stages ou dans des cadres bien specifiques ainsi que
l'amelioration du planning et de l'organisation des services de sante, des services d'education et
des services sociaux.

L6 ANSWER 54 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1999037669 PCTFULL ED 20020515
TITLE (ENGLISH): PEPTIDES WITH β1 INTEGRIN SUBUNIT DEPENDENT CELL ADHESION MODULATING ACTIVITY
TITLE (FRENCH): PEPTIDES POSSEDANT UNE ACTIVITE DE MODULATION DE L'ADHERENCE DES CELLULES DEPENDANT DE LA SOUS-UNITE D'INTEGRINE β
INVENTOR(S): McCARTHY, James, B.;
FURCHT, Leo, T.;
BRIENZO, Angela
PATENT ASSIGNEE(S): REGENTS OF THE UNIVERSITY OF MINNESOTA;
McCARTHY, James, B.;
FURCHT, Leo, T.;
BRIENZO, Angela
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9937669	**A1**	**19990729**

DESIGNATED STATES
W: CA JP US AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1999-US1236 A 19990121
PRIORITY INFO.: 1998-60/072,119 19980122
US 1998-60/072,119 19980122
US 1998-60/096,211 19980812
US 1998-60/096,211 19980812
US 1998-60/096,212 19980812
US 1998-60/096,212 19980812

ABEN Peptides capable of modulating β1 integrin subunit dependent cell adhesion which includes
a C-terminal aromatic amino acid residue and an amino acid residue having a lipophilic alkyl side
chain as the penultimate C-terminal residue are provided. These LipAr C-terminated peptides are
typically capable of modulating the β1 integrin subunit dependent adhesion of cells, such as
Ramos cells.
ABFR L'invention concerne des peptides capables de moduler l'adherence des cellules dependant de la
sous-unite d'integrine β qui comprennent un residu d'acides amines aromatiques a terminal C et
un residu d'acides amines possedant une chaine laterale d'alkyle lipophile se presentant comme un
dernier residu du terminal C. Ces peptides LipAr a terminaison C sont, en regle generale, capables
de moduler l'adherence des cellules dependant de la sous-unite d'integrine β, par exemple des
cellules de Ramos.

L6 ANSWER 55 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1998056408 PCTFULL ED 20020514
TITLE (ENGLISH): INHIBITORS OF MICROBIAL ADHERENCE OR INVASION AS THERAPEUTIC AGENTS AND BROAD-SPECTRUM ENHANCERS OF ANTIBIOTIC THERAPY

TITLE (FRENCH): INHIBITEURS D'ADHERENCE OU D'INVASION MICROBIENNE TENANT LIEU D'AGENTS THERAPEUTIQUES ET D'ACTIVATEURS A LARGE SPECTRE EN ANTIBIOTHERAPIE
INVENTOR(S): CLEARY, Paul, Patrick;
CUE, David, R.
PATENT ASSIGNEE(S): REGENTS OF THE UNIVERSITY OF MINNESOTA
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9856408	**A2**	**19981217**

DESIGNATED STATES
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW GH GM KE LS MW SD SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML MR NE SN TD TG

APPLICATION INFO.: WO 1998-US12019 A 19980610
PRIORITY INFO.: 1997-60/049,124 19970610
US 1997-60/049,124 19970610

ABEN The invention is directed to therapeutic use of compounds that function as inhibitors of microbial intracellular invasion of or adherence to host mammalian cells. Co-administration of the inhibitory compound with an antibiotic, such as penicillin, that inefficiently permeates mammalian cell membranes increases the efficacy of the antibiotic therapy.

ABFR L'invention concerne l'utilisation therapeutique de composes tenant lieu d'inhibiteurs d'invasion ou d'adherence microbienne intracellulaire dans des cellules mammaliennes hotes. En administrant conjointement le compose inhibiteur et un antibiotique (par exemple, de la penicilline) qui infiltre inefficacement les membranes des cellules mammaliennes, on ameliore l'efficacite de l'antibiotherapie.

L6 ANSWER 56 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1998033812 PCTFULL ED 20020514
TITLE (ENGLISH): MAST CELL PROTEASE PEPTIDE INHIBITORS
TITLE (FRENCH): INHIBITEURS PEPTIDIQUES DES PROTEASES DE MASTOCYTES
INVENTOR(S): STEVENS, Richard, L.;
HUANG, Chifu
PATENT ASSIGNEE(S): BRIGHAM AND WOMEN'S HOSPITAL, INC.
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9833812	**A1**	**19980806**

DESIGNATED STATES
W: CA JP AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE

APPLICATION INFO.: WO 1998-US1865 A 19980130
PRIORITY INFO.: 1997-60/037,090 19970205
US 1997-60/037,090 19970205

ABEN Compositions and methods for inhibiting a complex containing a mast cell protease are provided.
The compositions are useful for treating inflammatory disorders, such as

asthma, that are mediated
by release of a tryptase-6 protein. Methods for identifying additional specific inhibitors of a
complex containing tryptase-6 protein and a serglycin glycosaminoglycan also are provided.

ABFR La presente invention a trait a des compositions et des procedes destines a inhiber un complexe
renfermant une protease de mastocyte. Les compositions sont efficaces dans le traitement des
troubles inflammatoires tels que l'asthme grace a la liberation d'une proteine tryptase-6.
L'invention concerne egalement des procedes aidant a identifier les inhibiteurs specifiques
supplementaires d'un complexe contenant la proteine tryptase-6 et un serglycine glycosaminoglycane.

L6 ANSWER 57 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN

ACCESSION NUMBER:	1998025892 PCTFULL ED 20020514
TITLE (ENGLISH):	INTEGRIN ANTAGONISTS
TITLE (FRENCH):	ANTAGONISTES DE L'INTEGRINE
INVENTOR(S):	FISHER, Matthew, J.; FRANCISKOVICH, Jeffry, Bernard; JAKUBOWSKI, Joseph, A.; MASTERS, John, J.; SCARBOROUGH, Robert, M.; SMYTH, Mark; SU, Ting
PATENT ASSIGNEE(S):	ELI LILLY AND COMPANY; COR THERAPEUTICS, INC.; FISHER, Matthew, J.; FRANCISKOVICH, Jeffry, Bernard; JAKUBOWSKI, Joseph, A.; MASTERS, John, J.; SCARBOROUGH, Robert, M.; SMYTH, Mark; SU, Ting
LANGUAGE OF PUBL.:	English
DOCUMENT TYPE:	Patent
PATENT INFORMATION:	

NUMBER	KIND	DATE
WO 9825892	**A1**	**19980618**

DESIGNATED STATES
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW GH KE LS MW SD SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML MR NE SN TD TG

APPLICATION INFO.:	WO 1997-US22495	A	19971208
PRIORITY INFO.:	1996-8/762,117		19961209
	US 1996-8/762,117		19961209

ABEN Novel compounds, their salts and compositions related thereto having activity against mammalian
integrins are disclosed. The compounds are useful in vitro or in vivo for preventing or treating
thrombotic or restenotic disorders.

ABFR L'invention concerne de nouveaux composes, leurs sels et des compositions afferentes presentant
une activite contre des integrines mammiferes. Les composes sont utiles in vitro ou in vivo dans la

prevention ou le traitement de troubles de type trombo ou estenose.

L6 ANSWER 58 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1998024898 PCTFULL ED 20020514
TITLE (ENGLISH): THERAPEUTIC COMPOSITION COMPRISING THE KAL PROTEIN AND USE OF THE KAL PROTEIN FOR THE TREATMENT OF RETINAL, RENAL, NEURONAL AND NEURAL INJURY
TITLE (FRENCH): COMPOSITION THERAPEUTIQUE CONTENANT LA PROTEINE KAL ET UTILISATION DE LA PROTEINE KAL POUR LE TRAITEMENT DE LESIONS RETINIENNES, RENALES, NEURONALES ET NEURALES
INVENTOR(S): PETIT, Christine;
SOUSSI-YANICOSTAS, Nadia;
HARDELIN, Jean-Pierre;
SARAILH, Catherine;
ROUGON, Genevieve;
LEGOUIS, Renaud;
ARDOUIN, Olivier;
MAZIE, Jean-Claude
PATENT ASSIGNEE(S): INSTITUT PASTEUR;
CENTRE NATIONAL DE LA RECHERCHE SCIENTIFIQUE (CNRS);
PETIT, Christine;
SOUSSI-YANICOSTAS, Nadia;
HARDELIN, Jean-Pierre;
SARAILH, Catherine;
ROUGON, Genevieve;
LEGOUIS, Renaud;
ARDOUIN, Olivier;
MAZIE, Jean-Claude
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9824898	**A2**	**19980611**

DESIGNATED STATES
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW GH KE LS MW SD SZ UG ZW AM AZ BY KG KZ MD RU TJ TM AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML MR NE SN TD TG

APPLICATION INFO.:	WO 1997-EP6806	A	19971205
PRIORITY INFO.:	1996-8/761,136		19961206
	US 1996-8/761,136		19961206

ABEN KAL protein is identified the active agent in a therapeutic composition for treatment of injury to nerve tissue, including spinal cord tissue, as well as support of treatment for renal grafts. Additionally, therapeutic treatment of renal injury, and kidney transplantation and renal surgery, is effected by administration of KAL protein. The therapeutic agent may be administered locally, or intravenously. Retinal disorders may be similarly treated.

ABFR La proteine KAL est identifiee comme principe actif dans une composition therapeutique destinee au traitement de lesions du tissu nerveux, y compris de la moelle epiniere, et comme auxiliaire de traitement dans des transplantations renales. La proteine KAL est aussi administree dans le traitement therapeutique de lesions renales, greffes de rein ou en chirurgie renale. L'agent

therapeutique peut etre administre localement ou par voie intraveineuse. Des affections retiniennes
peuvent egalement etre traitees par ce procede.

L6 ANSWER 59 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1997041151 PCTFULL ED 20020514
TITLE (ENGLISH): CHOLINE BINDING PROTEINS FOR ANTI-PNEUMOCOCCAL VACCINES
TITLE (FRENCH): PROTEINES FIXANT LA CHOLINE POUR VACCINS ANTI-PNEUMOCOCCIIQUES
INVENTOR(S): MASURE, H., Robert;
ROSENOW, Carsten, I.;
TUOMANEN, Elaine;
WIZEMAN, Theresa, M.
PATENT ASSIGNEE(S): THE ROCKEFELLER UNIVERSITY
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9741151	**A2**	**19971106**

DESIGNATED STATES
W: AU CA FI JP MX AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1997-US7198 A 19970501
PRIORITY INFO.: 1996-8/642,250 19960501
US 1996-8/642,250 19960501
US 1996-60/016,632 19960501
US 1996-60/016,632 19960501

ABEN The invention relates to bacterial choline binding proteins (CBPs) which bind choline. Such
proteins are particularly desirable for vaccines against appropriate strains of Gram positive
bacteria, particularly streptococcus, and more particularly pneumococcus. Also provided are DNA
sequences encoding the bacterial choline binding proteins or fragment thereof, antibodies to the
bacterial choline binding proteins, pharmaceutical compositions comprising the bacterial choline
binding proteins, antibodies to the bacterial choline binding proteins suitable for use in passive
immunization, and small molecule inhibitors of choline binding protein mediated adhesion. Methods
for diagnosing the presence of the bacterial choline binding protein, or of the bacteria, are also
provided. In a specific embodiment, a streptococcal choline binding protein is an enolase, which
demonstrates strong affinity for fibronectin.

ABFR L'invention concerne des proteines bacteriennes fixant la choline (CBPs) qui fixe la choline.
De telles proteines sont particulierement utiles pour les vaccins contres les souches appropriees de
bacteries Gram positives, en particulier les streptocoques, et plus particulierement les
pneumocoques. L'invention decrit egalement des sequences d'ADN codant les proteines de fixation de
la choline bacteriennes ou un fragment de celles-ci, des anticorps contre des proteines de fixation
de la choline bacteriennes, des compositions pharmaceutiques comprenant des proteines bacteriennes
de fixation de la choline, des anticorps contre les proteines bacteriennes de fixation de la choline
utiles dans l'immunisation passive, et des inhibiteurs de petites

molecules de l'adhesion induite
par les proteines de fixation de la choline. Des procedes permettant de diagnostiquer la presence
d'une proteine de fixation de choline bacterienne, ou des bacteries, sont egalement decrits. Dans un
mode specifique de realisation, une proteine de fixation de choline streptococcique est une enolase,
ce qui demontre une forte affinite pour la fibronectine.

L6 ANSWER 60 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1997040072 PCTFULL ED 20020514
TITLE (ENGLISH): ADAM PROTEINS AND USES THEREOF
TITLE (FRENCH): PROTEINES ADAM ET LEUR UTILISATION
INVENTOR(S): CROUCHER, Peter, Ian;
MCKIE, Norman;
RUSSELL, Robert, Graham, Goodwin
PATENT ASSIGNEE(S): THE UNIVERSITY OF SHEFFIELD;
CROUCHER, Peter, Ian;
MCKIE, Norman;
RUSSELL, Robert, Graham, Goodwin
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9740072	**A2**	**19971030**

DESIGNATED STATES
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN YU GH KE LS MW SD SZ UG AM AZ BY KG KZ MD RU TJ TM AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML MR NE SN TD TG

APPLICATION INFO.:	WO 1997-GB1067	A	19970416
PRIORITY INFO.:	1996-9608130.2		19960419
	GB 1996-9608130.2		19960419

ABEN Described are ADAM 12 proteins, species variants, homologues, allelic forms, mutant forms,
derivatives, muteins and equivalents thereof, and various individual domains of ADAM 12 proteins,
various domain combinations, inhibitors thereof and various forms of therapy, diagnosis and
prophylaxis based thereon. Various therapeutic, diagnostic and prophylatic applications of proteins
of the ADAM (reprolysin) family in general, as well as their individual domains, domain
combinations, inhibitors and other materials based thereon, are also described.

ABFR La presente invention concerne des proteines ADAM 12, des variantes de l'espece, des
homologues, des formes alleles, des formes mutantes, des derives, des muteines et certains de leurs
equivalents. L'invention concerne egalement differents domaines specifiques des proteines ADAM 12,
differentes combinaisons de domaines, certains de leurs inhibiteurs et differentes formes de
therapies, de diagnostics et de prophylaxies ayant recours a ces domaines. L'invention concerne en
outre differentes applications therapeutiques, diagnostiques et prophylactiques des proteines de la
famille ADAM (reprolysine) en general, ainsi que leurs differents

domaines, combinaisons de
domaines, inhibiteurs et autres materiels les faisant intervenir.

L6 ANSWER 61 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1997035872 PCTFULL ED 20020514
TITLE (ENGLISH): CASPR/p190, A FUNCTIONAL LIGAND FOR RPTP-BETA AND THE AXONAL CELL RECOGNITION MOLECULE CONTACTIN
TITLE (FRENCH): CASPR/p190, LIGAND FONCTIONNEL DU RPTP-BETA ET DE LA CONTACTINE, MOLECULE DE RECONNAISSANCE DES CELLULES AXONALES
INVENTOR(S): PELES, Elior
PATENT ASSIGNEE(S): SUGEN, INC.
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9735872	**A1**	**19971002**

DESIGNATED STATES
W: CA JP AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1997-US5270 A 19970327
PRIORITY INFO.: 1996-60/014,199 19960327
US 1996-60/014,199 19960327
US 1997-8/826,134 19970326
US 1997-8/826,134 19970326

ABEN The 190 kDa Contactin ASsociated PRotein (CASPR/p190) is identified and is implicated as the
bridge between contactin and intracellular second messenger systems for the signal caused by the
binding of the carboxy anhydrase domain of RPTP'beta' to contactin and resulting in neurite growth,
differentiation or survival. Mammalian CASPR/p190 cDNAs and proteins are described, including those
from human and rat. In addition, particular domains of the proteins are characterized.
ABFR L'invention concerne l'identification de la proteine de 190 kDa associee a la contactine
(CASPR/p190). Cette proteine est responsable de la formation d'un pont entre la contactine et les
systemes messagers secondaires intracellulaires transmettant le signal qui est produit par la
liaison du domaine carboxy-anhydrase de la tyrosine-phosphatase de type recepteur (RPTP'beta') a la
contactine, et qui entraine la croissance, la differenciation et la survie des axones. L'invention
decrit les ADNc de la CASPR/p190 et les proteines des mammiferes, notamment celles provenant de
l'homme et du rat. Elle decrit egalement des domaines particuliers desdites proteines.

L6 ANSWER 62 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1995006660 PCTFULL ED 20020514
TITLE (ENGLISH): EPILIGRIN, AN EPITHELIAL LIGAND FOR INTEGRINS
TITLE (FRENCH): EPILIGRINE, LIGAND EPITHELIAL POUR LES INTEGRINES
INVENTOR(S): CARTER, William, G.;
GIL, Susana, G.;
RYAN, Maureen, C.
PATENT ASSIGNEE(S): FRED HUTCHINSON CANCER RESEARCH CENTER;
CARTER, William, G.;
GIL, Susana, G.;
RYAN, Maureen, C.

DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9506660	**A1**	**19950309**

DESIGNATED STATES
W: AM AT AU BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE HU JP KE KG KP KR KZ LK LR LT LU LV MD MG MN MW NL NO NZ PL PT RO RU SD SE SI SK TJ TT UA US UZ VN KE MW SD AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE BF BJ CF CG CI CM GA GN ML MR NE SN TD TG

APPLICATION INFO.: WO 1994-US10261 A 19940902
PRIORITY INFO.: 1993-8/115,918 19930902
US 1993-8/115,918 19930902

ABEN Nucleic acid sequences are disclosed encoding an E170 epithelial ligand and capable of hybridizing under stringent conditions to the nucleotide sequences derived from cDNA clones shown in the figure. Also disclosed are vectors containing the nucleic acid sequences, and cells transformed with the vectors. Methods are given for purifying and utilizing epiligrin, an epithelial glycoprotein complex, and its component glycoproteins, and for raising antibodies against components of this complex. Assay methods are further provided for identification of functional epiligrin in tissues.

ABFR Sequences d'acide nucleique codant un ligand epithelial E170 et capable de s'hybrider dans des conditions strictes aux sequences nucleotidiques derivees de clones d'ADNc illustres dans la figure. L'invention se rapporte egalement a des vecteurs contenant les sequences d'acide nucleique, et a des cellules transformees au moyen de ces vecteurs. Des procedes de purification et d'utilisation d'epiligrine, un complexe de glycoproteines epitheliales, et de ses glycoproteines constitutives sont decrits, ainsi que des procedes de developpement d'anticorps diriges contre les constituants de ce complexe. Des methodes de dosage destinees a l'identification d'epiligrine fonctionnelle dans des tissus sont egalement decrites.

L6 ANSWER 63 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1994011400 PCTFULL ED 20020513
TITLE (ENGLISH): PEPTIDES FROM HUMAN ICAM-2 AND FROM HUMAN ICAM-1 AND THEIR ANALOGS FOR USE IN THERAPY AND DIAGNOSIS
TITLE (FRENCH): PEPTIDES DE ICAM-2 ET ICAM-1 CHEZ L'HOMME ET LEURS ANALOGUES S'UTILISANT EN THERAPIE ET DIAGNOSTIC
INVENTOR(S): GAHMBERG, Carl, G.;
NORTAMO, Pekka;
LI, Rui
PATENT ASSIGNEE(S): HELSINKI UNIVERSITY LICENSING LTD. OY
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9411400	**A1**	**19940526**

DESIGNATED STATES
W: AU BB BG BR BY CA CZ FI HU JP KP KR KZ LK LV MG MN MW NO NZ PL RO RU SD SK UA VN AT BE CH DE DK ES FR GB GR

IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1993-FI480 A 19931115
PRIORITY INFO.: 1992-977,699 19921118
US 1992-977,699 19921118
US 1993-131,976 19931008
US 1993-131,976 19931008

ABEN ICAM-1 and ICAM-2 peptides and analogs are disclosed which are useful in preventing aggregation or adhesion of leukocytes or lymphocytes to endothelial cells. Such peptides and analogs may also be used to enhance the activity of leukocytes to target cells. Prevention of aggregation or adhesion of leukocytes or lymphocytes aids in the prevention of undesired immune responses, such as transplant rejection.

ABFR L'invention concerne des peptides et des analogues de ICAM-1 et ICAM-2 efficaces dans la prevention de l'aggregation ou de l'adhesion de leucocytes ou de lymphocytes a des cellules endotheliales. Ces peptides et analogues peuvent egalement s'utiliser pour amplifier l'activite de leucocytes vers des cellules cibles. La prevention de l'aggregation ou de l'adhesion de leucocytes ou de lymphocytes contribue a la prevention de reponses immunes indesirables, telles que le rejet de transplantations.

L6 ANSWER 64 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1993023526 PCTFULL ED 20020513
TITLE (ENGLISH): A NOVEL RECEPTOR FOR alpha4 INTEGRINS AND METHODS BASED THEREON
TITLE (FRENCH): NOUVEAU RECEPTEUR D'INTEGRINES alpha4 ET PROCEDES BASES SUR CELUI-CI
INVENTOR(S): VONDERHEIDE, Robert, H.;
SPRINGER, Timothy, A.
PATENT ASSIGNEE(S): CENTER FOR BLOOD RESEARCH, INC.
LANGUAGE OF PUBL.: German
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9323526	**A1**	**19931125**

DESIGNATED STATES
W: AU CA JP AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1993-US4956 A 19930521
PRIORITY INFO.: 1992-886,992 19920521
US 1992-886,992 19920521

ABEN The present invention is directed to a novel receptor for alpha4 integrins such as VLA-4, that is distinct from VCAM-1 and from fibronectin. Isolated nucleic acids encoding the receptor and antibodies to the receptor are also provided. The invention is also directed to pharmaceutical compositions, and methods of treating disorders involving an undesirable inflammatory or immune response by administering the VLA-4 receptor of the invention.

ABFR L'invention concerne un nouveau recepteur d'integrines alpha4 telle que VLA-4, laquelle est distincte de VCAM-1 et de la fibronectine. L'invention concerne egalement des acides nucleiques isoles codant le recepteur et des anticorps contre ledit recepteur. De

plus, l'invention concerne
des compositions pharmaceutiques ainsi que des methodes de traitement de troubles impliquant une
reponse inflammatoire ou immune indesirable, consistant en
l'administration du recepteur de VLA-4 de
l'invention.

L6 ANSWER 65 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1993011229 PCTFULL ED 20020513
TITLE (ENGLISH): NOVEL POLYPEPTIDES FOR PROMOTING CELL ATTACHMENT
TITLE (FRENCH): NOUVEAUX POLYPEPTIDES PROMOTEURS DE LA FIXATION DE CELLULES
INVENTOR(S): GINSBERG, Mark, H.;
PLOW, Edward, F.;
BOWDITCH, Ronald
PATENT ASSIGNEE(S): THE SCRIPPS RESEARCH INSTITUTE
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9311229	**A1**	**19930610**

DESIGNATED STATES
W: AU CA FI JP NO AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
APPLICATION INFO.: WO 1992-US10511 A 19921204
PRIORITY INFO.: 1991-804,224 19911205
US 1991-804,224 19911205

ABEN Novel polypeptides derived from human **fibronectin**, and fusion proteins containing those peptide
sequences are described which define a receptor binding site on **fibronectin** that binds to the
platelet receptor glycoprotein GPIIb-IIIa expressed by cells. The receptor binding site of human
fibronectin includes at least **fibronectin** amino acid
residues 1410-1436. The polypeptides facilitate
attachment of cells to substrates either alone or in conjunction with RGD-containing peptides.
Vectors preparing the fusion proteins and antibodies are also described. Methods for promoting cell
attachment and for **inhibiting cell adhesion**
are also described.

ABFR De nouveaux polypeptides derives de la fibronectine humaine et des proteines de fusion qui
contiennent ces sequences peptidiques definissent un site de liaison des recepteurs sur la
fibronectine qui se lie a la glycoproteine GPIIb-IIIa recepteur de plaquettes exprimee par les
cellules. Le site de liaison des recepteurs de la fibronectine humaine contient au moins les residus
1410-1436 des acides amines de la fibronectine. Ces polypeptides facilitent la fixation de cellules
sur des substrats, qu'ils soient utilises seul ou en association avec des peptides contenant des
sequences RGD. L'invention concerne en outre des vecteurs utiles pour preparer ces proteines de
fusion et ces anticorps, ainsi que des procedes de promotion de la fixation de cellules et
d'inhibition de l'adhesion de cellules.

L6 ANSWER 66 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1990013300 PCTFULL ED 20020513

TITLE (ENGLISH): ENDOTHELIAL CELL-LEUKOCYTE ADHESION MOLECULES (ELAMs) AND MOLECULES INVOLVED IN LEUKOCYTE ADHESION (MILAs)

TITLE (FRENCH): MOLECULES D'ADHESION LEUCOCYTES-CELLULES ENDOTHELIALES (ELAM) ET MOLECULES IMPLIQUEES DANS L'ADHESION DES LEUCOCYTES (MILA)

INVENTOR(S): HESSION, Catherine, R.;
LOBB, Roy, R.;
GOELZ, Susan, E.;
OSBORN, Laurelee;
BENJAMIN, Christopher, D.;
ROSA, Margaret, D.

PATENT ASSIGNEE(S): BIOGEN, INC.;
HESSION, Catherine, R.;
LOBB, Roy, R.;
GOELZ, Susan, E.;
OSBORN, Laurelee;
BENJAMIN, Christopher, D.;
ROSA, Margaret, D.

LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9013300	**A1**	**19901115**

DESIGNATED STATES
W: AT AU BE CA CH DE DK ES FR GB IT JP KR LU NL NO SE US

APPLICATION INFO.: WO 1990-US2357 A 19900427
PRIORITY INFO.: 1989-345,151 19890428
US 1989-345,151 19890428
US 1989-359,516 19890601
US 1989-359,516 19890601
US 1989-452,675 19891218
US 1989-452,675 19891218

ABEN DNA sequences encoding endothelial cell-leukocyte adhesion molecules ELAMs, methods for producing such molecules, and ELAMs (including the specific molecules ELAM1 and VLAM1 and 1b) essentially free of normally associated animal proteins are disclosed. Antibodies to ELAMs are also disclosed. DNA sequences encoding molecules involved in leukocyte adhesion (MILAs), method for producing such molecules and MILAs (including the specific molecule, CDX) essentially free of normally associated animal proteins are also disclosed. Antibody preparations which are reactive for MILAs are also disclosed. Methods for identifying molecules which inhibit binding of leukocytes to endothelial cells, methods for inhibiting leukocyte binding to endothelial cells, and methods for detecting acute inflammation are disclosed.

ABFR La presente invention decrit les sequences d'ADN encodant les molecules d'adhesion leucocytes-cellules endotheliales (ELAM), les methodes de production de ces molecules, et les ELAM (y compris les molecules specifiques ELAM1 et VLAM1 et 1b) essentiellement libres des proteines animales normalement associees. Sont aussi decrits les anticorps des molecules ELAM. La presente invention decrit en outre les sequences d'ADN encodant les molecules impliquees dans l'adhesion des leucocytes (MILA) et les methodes de production de ces molecules et MILA (y compris la molecule

specifique CDX) essentiellement libes des proteines animales normalement associees. Elle decrit
aussi les preparations d'anticorps qui reagissent aux MILA. Elle decrit encore les methodes
d'identification des molecules inhibitrices de la liaison des leucocytes aux cellules endotheliales,
les methodes d'inhibition de la liaison des leucocytes aux cellules endotheliales, et les methodes
de detection d'inflammation aiguee.

L6 ANSWER 67 OF 67 PCTFULL COPYRIGHT 2005 Univentio on STN
ACCESSION NUMBER: 1989011273 PCTFULL ED 20020513
TITLE (ENGLISH): ADHESION RECEPTOR FOR LAMININ AND ITS USE
TITLE (FRENCH): RECEPTEUR D'ADHESION POUR LAMININE ET SON UTILISATION
INVENTOR(S): RUOSLAHTI, Erkki, I.;
ENGVALL, Eva;
GEHLSEN, Kurt, R.
PATENT ASSIGNEE(S): LA JOLLA CANCER RESEARCH FOUNDATION
LANGUAGE OF PUBL.: English
DOCUMENT TYPE: Patent
PATENT INFORMATION:

NUMBER	KIND	DATE
WO 8911273	**A1**	**19891130**

DESIGNATED STATES
W: AT AU BE CH DE FR GB IT JP LU NL SE
APPLICATION INFO.: WO 1989-US2240 A 19890522
PRIORITY INFO.: 1988-196,986 19880520
US 1988-196,986 19880520

ABEN An adhesion receptor for laminin is provided. The receptor is isolated from cell or tissue
extracts and fractionated on an affinity column composed of cell attachment-promoting fragments of
laminin coupled to Sepharose in the presence of divalent cations. This receptor can be used to
prepare specific antibodies for the analysis of the amount of laminin receptor expressed by cells
and has other applications in cellular and tumor biology.
ABFR L'invention concerne un recepteur d'adhesion pour laminine. On isole le recepteur a partir
d'extraits cellulaires ou tissulaires, et on le fractionne dans une colonne d'affinite composee de
fragments de laminine favorisant la fixation de cellules, couples a du Sepharose en presence de
cations divalents. On peut utiliser ce recepteur pour preparer des anticorps specifiques afin
d'analyser la quantite de recepteur de laminine exprime par des cellules. Ce recepteur a d'autres
applications en biologie cellulaire et tumorale.